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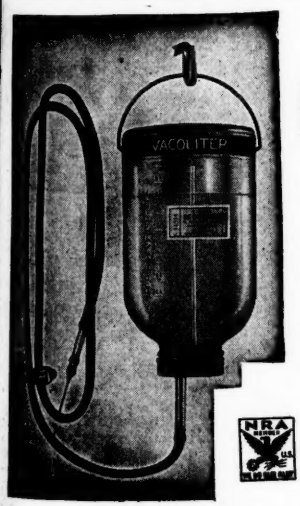

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CLINICAL INTERPRETATION OF THE ELECTROCARDIOGRAM*

BY LEWIS M. HURXTHAL, M.D.†

SINCE the introduction of the electrocardiograph by Einthoven in 1904, clinical electrocardiography has come into general use and today is accepted as an important aid in the diagnosis and treatment of heart disease. When employed as a method of registration of the heart beat for the purpose of classification and study of heart disease there can be no question that it is, and has been, of great value. But is our enthusiasm for its use in the practice of medicine justified? Is it as valuable and dependable as is commonly supposed? In answer to these questions it is not my purpose to appear to discourage the use of the electrocardiograph, but to emphasize the importance of caution when it comes to interpretation of electrocardiographic changes, particularly in relation to diagnosis and prognosis.

The electrocardiograph registers electrical changes which take place in the heart when it beats. These changes are recorded in the electrocardiogram by deflections or waves which depart from a given base line, so that each heart beat is shown by a series of these waves occurring at regular intervals, in case the heart is beating normally, and at irregular intervals, if the heart is beating irregularly.

In relation to electrocardiography, the heart may be conveniently divided into three parts:

1. The auricles.
2. The conducting apparatus.
3. The ventricles.

The conducting mechanism may be most simply described as a bundle of nervelike tissue arising in the auricle, extending down to the ventricles, where it divides into the bundle branches. By this conducting apparatus the wave of excitation is conveyed from the auricle to the ventricle.

Correspondingly, the electrocardiographic record of the heart beat may be separated into three parts:

1. The P wave, the result of the auricular beat.
2. The QRS waves, the result of the passage of the excitation wave through the conducting apparatus.
3. The T wave, the result of the ventricular beat.

Thus, without the contraction of the auricles there can be no auricular or P wave; without participation of the conducting mechanism the normal QRS group cannot be recorded; and likewise, without a contraction of the ventricles no T wave is registered. These waves as a rule can be easily identified because they have characteristic sizes and shapes. Only when the heart beat is abnormal do they change their relative form or position. The valves of the heart are not connected with the intrinsic mechanism of the heart beat and therefore have no direct influence on the electrocardiogram.

The electrical changes in the heart which occur as the result of the heart beat are transmitted to all parts of the body. As a matter of custom and practice, this electrical disturbance in the body is tapped at three definite points on the body surface; hence, the origin of the terms, the three leads or derivations. A slightly different picture is recorded in each of the three leads so that much more information is obtained by following this practice.

Abnormal Changes in the Mechanism of the Heart Beat. The chief components of the heart beat and the electrocardiogram have now been mentioned. Let us now consider the various changes that can take place in the mechanism of the heart beat. Generally speaking, these variations, too, may be grouped into three divisions:

1. Irregularities or disturbance of rhythm.
2. Heart block or disturbance in conduction.
3. Myocardial or change in muscular function.

1. *Disturbance of Rhythm.* In regard to disturbances of rhythm, the mechanism of the various irregularities will not be discussed here. It should be pointed out that the electrocardiogram serves admirably in identifying these abnormalities and at times provides the only available method of doing so. The two most common irregularities, namely, extrasystoles and auricular fibrillation, can be diagnosed clinically in the majority of instances. In such a capacity the electrocardiogram adds little to our usual methods of examination. In the rarer disturbance of rhythm, such as auricular flutter and paroxysmal tachycardia, the electrocardiogram serves a useful purpose in the differentiation of these conditions and simple acceleration

*From the Department of Medicine, Lahey Clinic, Boston.
Clinical Lecture at the American Medical Association Meeting, Milwaukee, June 13, 1933.

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of the heart. In hospital practice, particularly after surgical operations, there are times when the employment of the electrocardiogram is almost indispensable for the proper handling of cases wherein such abnormalities occur.

2. *Disturbance of Conduction.* Considering now the second group, disturbances of conduction, the electrocardiogram is a necessity in the correct diagnosis of the various types of heart block. From the clinical point of view, block occurring at the bundle of His is the most familiar one. In this form of heart block we are mostly concerned with the question: Does the patient have symptoms from this condition? It is well known that this type of heart block can be present for years without causing any circulatory disturbance. Thus, in these cases, when the rest of the electrocardiographic picture is normal, we learn little aside from identification by the electrocardiographic registration of the block alone.

To anyone who has a working knowledge of electrocardiography the irregularities and heart block of the above-mentioned type are as a rule easily recognized and therefore require little interpretation.

The most significant errors in electrocardiography occur as the result of misinterpretation of changes in the size, shape, and direction of the waves. More prognostic significance is attached to these types of changes than to changes in rhythm or conduction in the upper part of the conducting apparatus. It is this aspect of electrocardiography which, it seems to me, requires the greatest consideration from the clinical point of view.

CHANGES IN THE SIZE, SHAPE, AND DIRECTION OF THE QRS AND T WAVES

We now come to the last group in which changes in the size, shape, and directions of the electrocardiographic waves take place. These can be limited for practical purposes to the QRS group and the T wave. These are essentially ventricular events. What are the types of changes which they may undergo? It would be well first to review in a general way the types of changes which can take place in the ventricles themselves and then correlate them with the known electrocardiographic variations. Broadly speaking, these are the following:

1. Variations in the force of the heart beat.
2. Variations in the speed of the spread of the excitation wave and in the rapidity of beating.
3. Change in the position of the heart in the body or of the relative size of the heart or its chambers.

These gross variations in the heart or its position are reflected in the electrocardiogram. Correspondingly, the changes which they produce in the electrocardiogram are, generally speaking, as follows:

1. Variations in the height of the waves.
2. Variations in the width of the waves.
3. Variations in the direction of the waves.

As a well-marked example of the correlation between the features of the electrocardiogram and the gross variations which are listed above, take for instance hyperthyroidism and hypothyroidism or myxedema. Here it is well known that the force of the heart beat is accentuated in hyperthyroidism and diminished in myxedema. In well-marked cases of myxedema, low waves are found, whereas in hyperthyroidism, uncomplicated by other heart complications, exaggerated waves may be found.

As an example of widening of the waves, any lesion which delays the spread of the excitation wave, such as is found with a block of one or the other bundle branches, will cause marked widening of the QRS group. Such a block is most commonly found as the result of arteriosclerotic degenerative changes in the heart.

Change in the position of the heart can most strikingly be shown when electrocardiograms are taken in individuals with dextrocardia. Here the waves in the electrocardiogram are directly opposite to those found in people with hearts in the usual position.

These illustrations of the more pronounced changes are mentioned for the purpose of demonstrating contrasting conditions and their effects on the electrocardiogram. With these extremes in mind, we can now consider some of the isolated abnormalities which constitute the main subject of this paper. Before doing this, however, it might be well to present a review of some of the more common agents which may cause changes in the ventricles. These may be listed under several headings, as follows:

1. Infection, local or general—rheumatic fever, diphtheria, streptococcus infections, syphilis.
2. Toxic agents—drugs, especially digitalis and quinidine, uremia, carbon monoxide.
3. Degenerative disease, especially coronary arteriosclerosis.
4. Mechanical, such as wounds, tumors, intrathoracic lesions, causing position changes.

These agents affect the heart in different ways, some causing more or less characteristic electrocardiographic patterns. These will now be taken up in detail.

VARIATIONS IN THE QRS GROUP

Let us first examine the QRS group and its variation in size. Normally the greatest deflection of this group should not measure less than 7 to 8 millimeters, nor more than 20 millime-

ters. Although some latitude may be allowed in this normal range, measurements which exceed these limits are usually an indication of some abnormality. Most frequently high excursions of the QRS are found in hypertension, where there is gross hypertrophy of the left ventricle. Their presence adds little if any diagnostic or prognostic evidence to that obtained from the history, physical, and other methods of examination.

LOW WAVES

Low QRS waves are frequently called low complexes, or complexes with low electromotive potential. What is the clinical significance of the finding of low QRS waves? This is indeed a difficult question to answer. One must of course know what disease, if any, is afflicting the patient. The presence of myxedema would naturally explain the finding of low complexes. Here again the prognostic significance is that of the disease rather than that of the electrocardiographic change. Other conditions in which there is a low general functional state of the entire body might also give rise to low waves. Or, as it has been stated, low waves may be registered in patients with emphysema due to mechanical interference. Not infrequently, low waves are found in patients with congestive heart failure, but it is doubtful if much significance should be attached to these findings in such cases, first, because they may improve with the clearance of congestive heart failure, and secondly, the only criterion so far as prognosis is concerned is whether the patient can recover and carry on. And in cases of this kind there is no reliable sign by which we can predict the immediate outcome. From the statistical point of view, the prognosis of low QRS waves, as with some other electrocardiographic abnormalities, is more or less unfavorable. However, statistical studies are often made from groups with cardiovascular complaints so that the true prognostic import as often reported may be misleading.

When low complexes are combined with one or two other electrocardiographic abnormalities, which are on the whole regarded as evidence of myocardial damage, the entire electrocardiographic picture is probably of more prognostic significance than when only one of these variations alone is present. For example, if one were to find low complexes, heart block, and auricular fibrillation present in one tracing, the chances are that the patient would be found to have very little myocardial reserve and the prognosis indeed would be unfavorable.

WIDTH OF QRS GROUP

Next to be considered is the duration, span, or width of the QRS group of waves. This group is an important one to note. To a slight but unimportant degree the width may vary with the heart rate. In a normally functioning heart, the duration of this complex should not exceed .11 of a second. A slight degree of slurring, as it is commonly called, or prolongation of these complexes is not uncommon and while this may be considered evidence of heart damage either focal or general, little importance can be attached, particularly if the total duration of the QRS group is not abnormal.

It has been recently suggested that insurance applicants who show a QRS interval of over .11 of a second should be considered as greater risks. Only time can tell whether this is a justifiable criterion. It need not concern us particularly here, but it would be well to point out that while this group as a whole may show a higher mortality rate, we are not justified in making an individual clinical diagnosis on this basis alone nor of informing the patient that he has a heart disease.

BUNDLE BRANCH BLOCK

A greater degree of widening is found in bundle branch block. The QRS group here is markedly prolonged, usually over .15 of a second. Opposite directions of the main deflections of the QRS group are found in Leads I and III in this condition, and as a rule the T waves point in the opposite direction of the QRS complex. When the right bundle branch is affected the main deflection of the QRS group points downward in Lead I, whereas the reverse holds true when the left bundle branch is affected.

Bundle branch block is a finding of importance and is frequently associated with grave cardiac damage. Yet it can be readily understood that it would require only a minute lesion to produce this abnormality. Furthermore, it has been found in some cases to be purely functional and to be brought about by vagal stimulation. Functional bundle branch block with a short P-R interval occurring in subjects with paroxysmal tachycardia in young people was first described by Wolf, White, and Parkinson. Their patients were otherwise quite healthy and showed no evidence of serious heart disease.

Bundle branch block may also occur during acute febrile diseases and while most of the time the lesion may remain permanent, nevertheless, in a goodly number, the block may disappear after the infection subsides. (Place, Pardee.) At this point it might be well to remind you that it is a curious and interesting fact that certain diseases affect one part of the heart more than another. Thus diphtheria is particularly apt to produce heart block either of the bundle of His or the bundle branches. Rheumatic fever is particularly prone to produce a prolongation in the interval between the P and R waves, due to a partial or complete block in the auricular ventricular node or bundle of His, but such an attack by this disease upon the bundle branches rarely if ever hap-

pens. Other acute diseases may attack the bundle branches, but their results are most likely to be shown by changes in the T waves. It is a debatable point whether the toxicity of hyperthyroidism affects the heart in a structural way but there is no question that it affects it in a functional way. Auricular fibrillation is a common result of hyperthyroidism, as well as minor changes in the T waves, but heart block and lesions of the bundle branches are perhaps never produced by hyperthyroidism itself.

While the finding of bundle branch block may make one more watchful during an acute infection and make us feel more uncertain as to the outcome, nevertheless, in view of the fact that it may disappear and if the heart shows no evidence of failing, we should not be greatly concerned over the electrocardiographic records.

Bundle branch block may be found in patients with disease of the coronary arteries, with or without anginal symptoms. It may also occur as the result of acute coronary thrombosis. The finding of bundle branch block in angina pectoris might be considered on first thought to be of grave prognostic significance, yet in a recent survey by White and Bland on the prognosis of angina pectoris and coronary thrombosis in over seven hundred patients, it was not found to have any prognostic value. In other words, the prognosis was the outlook of the disease in general and not the result of any one finding in the various types of examination. Their follow-up actually showed a greater number with bundle branch block in that group which lived over seven years after the onset of the disease than those who died within three years after the onset of the disease.

But if we consider, then, this finding alone without knowledge of the physical examination and history, as it might be from the insurance point of view, we are forced to admit that in general it indicates serious changes in the myocardium and that the prognosis must be guarded. On the other hand, as glycosuria does not always mean diabetes and albuminuria does not always signify Bright's disease, in the same manner bundle branch block does not always signify serious heart disease.

THE "Q" WAVE

Another feature of the QRS group to be considered is the so-called Q wave. Attention has been directed to the Q wave of Lead III during the past few years. It is said to be of importance when it points downward in Lead III and the total deflection is 25 per cent or more of the greatest excursion of the QRS group in the other leads. The finding of such a Q wave has been thought to be a fairly frequent finding in coronary artery disease. A warning should be broadcast in regard to the correct identification of this phenomena. It would seem that what is frequently called a prominent Q wave in Lead III is not that at all. It has been a common practice not to attempt to identify individually the various components of the QRS group. But since the appearance of the Q wave on the horizon of diagnostic signs, greater attention should be directed toward this end. I will not go into detail in regard to the possibilities of accomplishing this, but will state what seems to be important in regard to the identification of prominent Q waves of Lead III. First, what is commonly called a prominent Q wave in Lead III is in reality an inverted R wave, or a fusion of an inverted Q made more prominent by an inverted R wave. This appears to be true in my experience even when there is no initial upward wave. The Q wave of the various leads behaves in general like the R and S waves so that, as a rule, when there is a downward Q wave in Lead I, there is not likely to be a prominent down Q wave in Lead III. Secondly, where there appears to be no Q wave in Lead I, close inspection will often show the initial and upward part of the R wave to be slurred. This slurring is, I believe, a fusion of an upward Q wave with the R wave. In a case of this sort, the Q wave in Lead III might and should be inverted but the great depth is the result in most instances of an inverted R wave.

THE R-S-T INTERVAL

The next important observation to be made in the electrocardiogram is the interval between the S wave or R wave and the T wave. In interpreting the variations in the S-T or R-T interval, it is important to determine whether the patient has had digitalis. Digitalis is so commonly given for heart complaints of any type that unless this fact is kept constantly in mind, the history of taking it might be overlooked, and thus influence the interpretation of an electrocardiogram. Not that it would always make a great difference, but there are times when it is conceivable that even a gross error in diagnosis might be made because of this omission.

Normally, the S-T or R-T interval may not always coincide with the base line. There are a considerable number of variations in the origin, shape, and direction of the S-T interval, but they are as a rule slight. The greatest change in position of the S-T interval is found following acute coronary thrombosis. At times this interval may be as far away from the base line as the main deflection of the R or S wave. While this abnormality has been shown to occur in other conditions such as pericarditis, yet clinically it is not likely that pericarditis might be confused with coronary thrombosis although it is true that pericarditis might be present following coronary thrombosis. Thus, the history and the physical findings should be sufficient to make this an unimportant consideration. It is unusual for the abnormally placed R-T or S-T interval to remain in such a position. Even-

tually it subsides and is usually replaced by the so-called coronary T wave. This will be discussed a little later on.

VARIATIONS OF THE "T" WAVE

We can now take up the last wave in the electrocardiogram, the T wave. This wave is produced by the contraction of the ventricles and is thus entirely a record of muscular activity. The height of the T wave as well as its length is probably a summation of effects produced by the contraction of a great number of muscle fibres at slightly different intervals, the largest number contracting at the apex of the wave. The irritability or functional state of the myocardium is to a certain degree reflected in the T wave. Cooling the heart muscle either directly or by the drinking of water will produce a lowering or inversion at the T wave, while warming can cause an exaggeration of the normal amplitude.

Normally, the T wave is upright in Leads I and II and variable in Lead III. Although it is not definitely proved which ventricle plays the predominating part in the production of the T wave, in any one lead, pathological studies would seem to indicate that the left ventricle may be responsible for the T wave in Lead I and partially so for the T wave in Lead II. Likewise, the right ventricle is reflected in the T wave of Lead III and partially in the T wave in Lead II. It must be borne in mind that the T wave in all leads is the composite record of muscular activity in both ventricles and that activity in one ventricle opposes the activity in the other ventricle, because in general the direction of the excitation wave is flowing in the opposite direction. Thus, it might be said that the T wave in Lead I is normally upright because the total effects of the contraction of the left ventricle in this lead are greater than those of the right ventricle. It is difficult to prove this hypothesis; yet on such a basis with a few other considerations of which I shall speak later, it is said to be possible to predict with some degree of success the location of coronary infarcts by changes in the T waves.

Let us first consider variations in the height of the T wave. In apparently healthy people this variation is considerable. It is impossible to lay down any sharply defined limits, but any T wave under two millimeters in height can at least be classed as low. This rule must apply to the highest T wave in either Lead I or II. It does not hold for Lead III, the unstable lead.

The finding of the low T waves does not indicate any specific lesion; it merely indicates in a general way some alteration of function during the process of contraction. One must, therefore, know the details of the history, treatment, physical examination, and in certain cases, laboratory data. For example, it may be associated with malnutrition or general debility from any cause. It may be found during or following some toxic infection either local or systemic. It may occur practically in any form of heart disease. Thus, it really gives no information of any definite value. In certain instances, it represents the status at a given moment, and further variation would be found if records were taken at subsequent intervals. The practice of taking serial electrocardiograms, if electrocardiographic study is desired, cannot be too strongly recommended in any condition which, by virtue of its pathological physiology, does not remain constant. In so doing, one might obtain evidence, for example, of a slow coronary thrombosis or of the recovery from the effects of medication, such as digitalis.

When it comes to finding a very low or flat T wave, more attention is warranted. When this finding is associated with low amplitude of the QRS complex, its importance, as stated before, is thereby increased.

Inversion of the T wave demands careful consideration. This does not refer to the inverted T waves of premature beats or extrasystoles or those found in bundle branch block; these have an entirely different significance. As is the case with low or flat T waves, inversion may be the result of some general toxemia. Thus, when found under these conditions, the significance of inverted T waves is to a certain extent obvious. Unless extensive myocardial injury takes place, a return toward normal should take place in most acute infections, and also in myxedema if adequate treatment can be or is carried out.

The one drug which produces definite T wave inversion is digitalis. When thorough digitalization has been effected, the T waves will be found to be well inverted, and usually in a characteristic manner. The origin of the wave may be normal or depressed, after this the wave points sharply downward, at times almost vertical, it then rounds to a horizontal position, following which there is a gradual rise to the base line. Many times the curve is so typical that one can say without the least hesitation that a digitalis effect is responsible for the T wave inversion. Thus, it can be readily appreciated that here is a way in which digitalis therapy can be followed. It may be discovered occasionally by the electrocardiogram that digitalis, when administered by mouth, has not been absorbed and it may be subsequently demonstrated that this has been true through the effect of intravenous injection of digitalis, not only on the electrocardiogram but also on the clinical course. It also affords a method of determining, once digitalization has been carried out, if the daily ration prescribed is sufficient to maintain saturation.

During anginal seizures, the T wave may show transient changes. This is probably associated with a relative ischemia of the heart muscle as the result of coronary artery spasm.

It has also been shown that following a sudden drop in peripheral blood pressure, T wave changes as well as other abnormalities may take place. These, too, might be ascribed to ischemia as the result of a diminished coronary blood flow attendant upon the drop in blood pressure. Thus, one must be careful not to interpret too hastily changes of this nature following collapse as due to possible coronary thrombosis.

In clinical practice we are perhaps most interested in the T wave in connection with coronary disease and coronary thrombosis. Coronary sclerosis and narrowing can exist without symptoms or physical signs or electrocardiographic evidence. Theoretically, trouble does not appear until there is some interference with blood supply. This may come on slowly, working changes in the myocardium, or it may manifest itself only on exertion, or it may suddenly result following a sudden occlusion from thrombosis. It is well to remember that any of these forms of coronary occlusion may occur without pain. It is in this painless type that electrocardiographic methods of study may be particularly valuable, and at times indispensable. At this point we can return to the question of the use of the T waves in identifying the coronary artery involved.

The right coronary artery supplies the right ventricle and the posterior portion of the left ventricle, while the left coronary artery supplies the remainder of the left ventricle. Barnes and Whitten state that when the left coronary artery is occluded, the T wave in Lead I or Lead I and II is inverted. On the other hand, inversion of the T wave in Lead III or in Lead II and III is present when the right coronary artery is thrombosed.

During acute coronary thrombosis, if electrocardiograms are taken quite early, no changes at all may be recorded. In fact, as in experimental work, the T waves may be accentuated. Serial records are of great value in these cases, for shortly progressive changes will appear and be registered clearly in the electrocardiogram. Thus, if the diagnosis be in doubt, the electrocardiogram may give the first definite clue to the diagnosis. Subsequent tracings may show a prompt inversion of the T waves or there may be a fusion of the R or S wave as in the T wave. Fusion of these waves gives rise to high or low S-T interval which was mentioned before. This is particularly characteristic of acute coronary thrombosis. From this abnormality there usually evolves a series of changes which eventually give rise to an inverted T wave. Through this evolution there appears the upright T wave, with a high origin, then finally an inverted T wave not only with a high origin but also with a characteristic beginning which is opposite in form to that caused by digitalis. Instead of a sharp downward dip as is the case with the digitalis T wave, it runs horizontally for a short distance and then curves gradually downward producing a convex curve. Now, when this takes place in Lead I, as above described, the reverse usually takes place in Lead III, with intermediate changes in Lead II.

The recognition of any one of these different stages in the evaluation of the coronary T wave is at times of value. It not only identifies in many instances the nature of the process going on, but also is an indication for repeated tracings especially where there may be some doubt as to the diagnosis. While these changes may take place slowly in patients with angina and presumably with a slow coronary occlusion, they are more characteristic of acute thrombosis, a matter which can be decided upon by the acute nature of the patient's symptoms.

Inversion of the T waves following coronary thrombosis may disappear in a few months. This does not insure against the possibility of another attack and thus it cannot be interpreted as a favorable sign. One is tempted to mislead the patient when the electrocardiogram becomes normal under such circumstances. It is entirely unwarranted.

Another cause of T wave inversion as suggested by Barnes and Whitten is that due to strain produced upon one ventricle or the other. For example, in hypertension strain is produced upon the left ventricle, and in mitral stenosis strain is produced on the right ventricle. This constant strain produces a state of fatigue giving rise to physiological changes in the muscle which in turn cause T wave inversion. Thus, in left ventricular strain, inversion of the T wave in Lead I may occur, and in right ventricular strain, inversion of the T wave in Lead III, Lead II showing possible changes in either one or the other of the two conditions. At any rate, such T wave changes do occur and no coronary narrowing or disease can be found at postmortem to cause them. Diffuse disease of the myocardium may also cause T wave inversion; the cause of the inversion, however, may also be one of muscle fatigue rather than due to the actual structural changes. Not infrequently the T wave as well as other parts of the electrocardiographic picture may improve upon the relief of congestive heart failure, thus indicating relief of strain rather than any change in the underlying pathology.

The changes in the T wave with preponderance may not always be due to ventricular strain. T wave inversion is often found in axis deviation where ventricular strain does not appear to be present. T wave inversion is more often found in Lead I with left axis deviation and in Lead III or Lead II and III in right axis deviation. Inversion of the T waves in Lead II and III is not infrequently found in young people without any evidence of heart disease. As a rule, these individuals are of the ptotic build with the vertical type of heart. Since this type

of heart tends more to right axis deviation than normal, it would seem here that it is of no clinical significance as regards a possible strain on the right ventricle.

HEART DISEASE WITHOUT ELECTROCARDIOGRAPHIC CHANGES

Before closing, it must be pointed out that a patient may have serious heart disease of almost any type, and electrocardiographic findings may be entirely normal. Such findings do not deprecate the value of the electrocardiogram but should serve to remind us that the first requisite in a heart examination is a careful history which will reveal, for example, angina pectoris, and a thorough physical examination which will often give all needed information as to diagnosis and prognosis. In borderline cases, the electrocardiogram may be the deciding factor in diagnosis, as is often true with roentgenography.

CERTAIN ASPECTS OF BRONCHIECTASIS WITH SPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT*

BY FREDERICK T. LORD, M.D.†

MUCH progress has been made during the past decade in the diagnosis and treatment of bronchiectasis. This is due to advances in roentgenography and the use of profile films, investigation of the bronchial tree by means of an opaque medium and the development of thoracic surgery with increased safety in the performance of lobectomy. Recognition of bronchiectasis in its early stages makes it apparent that the disease as an independent affection is more common than was formerly appreciated.

CLINICAL ASPECTS OF BRONCHIECTASIS

Bronchiectasis usually arises insidiously. It may follow an acute and apparently simple bronchitis, or unresolved lobar or latent or obvious bronchopneumonia. Cough and expectoration are the principal and often the only significant symptoms and may occur in attacks with relative or complete freedom during the intervals. The sputum is mucopurulent and has an offensive odor in about one-half the cases. This is stale or musty and not ordinarily foul as with lung abscess or gangrene. Separation of the sputum into three layers on standing is not peculiar to bronchiectasis. Hemoptysis occurs in about one-half the cases. The blood is present in streaks or as moderate or profuse hemorrhage. On examination of patients with the purely bronchial form of the disease there may be no physical signs other than coarse, medium or fine, non-consonating râles. The râles are likely to be variable in character and occurrence and may disappear after evacuation of the cavities by cough. Persistent localization at one or several places may serve to suggest the site of the disease. Roentgen-ray examination with the cylindric form may show increased linear markings in the involved region and with the saccular ectases the presence of ring shadows with or without fluid level. The use of lipiodol is desirable to determine more accurately the presence and extent of dilated bronchi.

In well-established cases, restoration of dilated bronchi to normal cannot be expected, but the condition is not incompatible with a long term of life. There is, however, the danger of extension of infection to nearby or remote parts of the lungs or elsewhere with resulting bronchopneumonia, abscess, gangrene, empyema, cerebral abscess or meningitis. Hemoptysis may be fatal in a small proportion of cases.

BRONCHIECTASIS SIMULATING TUBERCULOSIS

The confusion of bronchiectasis with tuberculosis is likely to occur in incompletely investigated cases, and especially in those with hemoptysis. Of thirty-six cases in the Massachusetts General Hospital series, fourteen were admitted from sanatoria. Profuse hemoptysis occurred in twelve. It was an initial symptom in only one. This patient, a girl of sixteen with bronchiectasis of the left lower lobe, was apparently well until five months before admission when she coughed up a teaspoonful of blood. There were no preceding symptoms, but a "cold" one to two weeks later was followed by persistent cough and the expectoration daily of about six ounces of odorless, purulent sputum. Phrenic paralysis and the separation of adhesions on the left side has been done preliminary to lobectomy. In a second patient with bronchiectasis of the right lower lobe, repeated large hemoptyses menaced life. This patient, a woman of thirty-four, was in contact with a tuberculous father at the age of seven. Three years before admission she began to have pain in the region of the right costal margin starting to the right of the xiphoid and radiating about the right chest in the region of the costal margin to the back below the right shoulder blade. The gall bladder was explored in an outside hospital one and one-half years later and was found to be normal. Five days after operation she had an hemoptysis amounting to about a pint, with-

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out preceding cough. This was followed by frequently repeated hemorrhages. There was a bad odor with cough. She expectorated about one-half ounce of greenish, malodorous sputum daily. Artificial pneumothorax and repeated refills failed to stop the bleeding. As the anesthesia was started for the operation of lobectomy, a profuse hemoptysis occurred that ceased only when a tourniquet was applied to the right lower lobe, which after removal showed two dilated bronchi, each 1 cm. in diameter with walls 2 mm. thick. Microscopic examination showed cylindrical-celled epithelium and neighboring fibrosis with greatly dilated veins in apposition with the cavity. The veins were for the most part thrombosed. In several there were aneurysmal breaks in the walls.

In the differentiation of bronchiectasis and pulmonary tuberculosis, an offensive odor to the sputum, repeated failure to find tubercle bacilli in purulent specimens, x-ray evidence of dilated bronchi at the bases and absence of subapical involvement are of importance.

DISTINCTION BETWEEN BRONCHIECTASIS AND ABSCESS

Acute lung abscess is not likely to be confused with bronchiectasis. In the more chronic types of abscess there may be difficulty in the differentiation. There may be abscess in one place and bronchiectasis in another. The two conditions may be combined with dilatation of bronchi communicating with the abscess or ulceration of the wall of an ectatic bronchus. With abscess there is more often a history of onset following operation on the upper parts of the respiratory tract and the odor of the sputum is much more offensive. On x-ray examination lung abscess is likely to show as a single area of increased density at the periphery of the lung, with central radiance and fluid level, while dilated bronchi are usually multiple, centrally placed, without neighborhood involvement, unless complicated, and without fluid level. Lipiodol commonly fails to enter the abscess cavity, but bronchial dilatations are usually readily visualized by this means after evacuation by postural drainage in the absence of bronchostenosis. The distinction between saccular ectases in an indurated lung and multiple lung abscesses may be difficult or impossible.

RELATIVELY "DRY" BRONCHIECTASIS

Cases of "dry" bronchiectasis have been described with hemoptysis a prominent symptom. There are no such cases in this series, but in a number of instances, the amount of purulent sputum was small compared with the bleeding.

BRONCHOSCOPY

This is indispensable for the determination of the cause of bronchial obstruction. It may be helpful in determining the source of blood or pus. In our experience, the instillation of lipiodol into the trachea below the vocal cords after evacuation by postural drainage gives as satisfactory results as bronchoscopic introduction. With us the bronchoscope has not been more helpful in treatment than postural drainage in the absence of bronchostenosis.

FAILURE OF CONSERVATIVE MEASURES

Improvement of nutrition, change of climate, treatment of infected sinuses and tonsils, inhalations, intratracheal injections, endobronchial treatment other than removal of foreign bodies and vaccines have all proved disappointing. Postural drainage may diminish the amount of sputum, but cannot be expected to cure.

COLLAPSE THERAPY

The pathology of bronchiectasis limits the value of collapse therapy. Parts of the lung supplied by the affected bronchi are already atelectatic in a considerable proportion of cases. Complicating adhesive pleurisy, fibrosis of lung tissue and conversion of the walls of dilated bronchi into dense scar tissue with or without remnants of cartilage, interfere with collapse of the cavities. Persistence of the superficial layer of mucous membrane would prevent obliteration of ectases even if their walls could be brought together.

Artificial Pneumothorax. To judge from reports in the literature, improvement may be expected in a considerable proportion of cases. Complete, permanent recovery is, I think, doubtful. Lipiodol may show persistence of the bronchiectasis in spite of the collapse. Improvement may continue only while the pneumothorax is maintained and there is the danger of producing empyema by the rupture of pleural adhesions. In the Hospital series, the method was carried out and failed in four cases, three of whom later came to operation.

Phrenic Nerve Operation. Results of phrenic nerve paralysis reported in the literature have been apparently favorable in a small proportion of cases. Temporary phrenic nerve paralysis is desirable with unilateral, lower and middle lobe bronchiectasis as a preliminary to or at the time of lobectomy. A phrenic nerve operation was done in twenty-one cases. In four the phrenic nerve and the lung operation were done at the same time. In fourteen cases, an interval of four days to sixteen months elapsed between the phrenic nerve and the lung operation. Three of the fourteen were improved by the procedure, but radical operation, nevertheless, seemed desirable. There are three additional cases which have not yet come to radical operation, with improvement in two. There are, thus, five out

of seventeen with improvement following phrenic nerve operation, or about one in three cases.

Thoracoplasty. This may be considered for extensive, multilobar, unilateral bronchiectasis. Cure has been reported in the literature by this means in a small proportion of cases. Our experience is limited to its use in four cases, without favorable results and one death two and one-half months after operation.

Intrapleural Pneumolysis. Following the separation of adhesions and the walling off of the pleural cavity at the first-stage operation preliminary to lobectomy, the affected part of the lung, held by adhesions, may be free to contract with resulting diminution in size and in the amount of sputum. In two cases, the improvement has been so marked as to lead to postponement of the second-stage operation.

In our experience with collapse therapy, improvement may follow phrenic nerve paralysis and the first stage of lobectomy. Cure is to be expected in well-established cases only by removal of the dilated bronchi.

LOBECTOMY

Progress in the development of lobectomy and its application to bronchiectasis are due to such pioneers in this field as Lilienthal (*Ann. Surg.* 75:257, 1922) Graham (*Arch. Surg.* 6:321 [Jan.] 1923) Whittemore (*Ann. Surg.* 86:219 [Aug.] 1927) Brunn (*Arch. Surg.* 18[1]:490 [Jan.] 1929); (*Surg. Gynec. Obst.* 55:616 [Nov.] 1932) Shenstone (lecture delivered before the Brit. and Canad. M. A., Aug. 27, 1930), also Shenstone and Janes (*Canad. M. A. J.* 27:138 [Aug.] 1932) Alexander (*Surg. Gynec. Obst.* 56:658 [March] 1933) and Churchill (*J. Thoracic Surg.* 2:254 [Feb.] 1933); (*New Eng. J. Med.* 208:323 [Feb. 9] 1933).

Indications for Operation. In considering lobectomy, anteroposterior and lateral x-ray examination without and with lipiodol are desirable preliminaries to determine the character and extent of the lesions. Care must be exercised with respect to the presence of dilated bronchi in more than one lobe and unfortunately complete reliance cannot be placed on lipiodol to exclude multilobar involvement.

In one of Janes' (*Brit. J. Surg.* 21:257 [Oct.] 1933) cases, a cavity the size of a hen's egg in the upper lobe failed to fill with lipiodol and was found at autopsy. In one of our cases, two dilated bronchi in the right lower lobe, each about one centimeter in diameter, were missed by repeated x-ray examination after lipiodol insufflation.

Lobectomy is to be considered with chronic and incapacitating, unilateral bronchiectasis confined to the middle and the lower lobes. In making the decision regarding operation, the following additional considerations are important: The patient's age should not exceed 40-45. Operative interference should not be undertaken during an acute exacerbation of the disease. Poor general condition is a contraindication. Dependence of others on the patient's earning capacity may make operation undesirable on account of the risk.

Operative Mortality. In 212 cases of total or subtotal lobectomy collected by Ballon, Singer and Graham (*J. Thoracic Surg.* 1:502 [June] 1932) there were seventy-two (34%) operative deaths. The hazard of operation has steadily diminished with a more careful selection of cases, increasing surgical experience and improvement in technic. This is illustrated in the experience at the Massachusetts General Hospital, where twenty-nine cases have been subjected to lobectomy. It is not my purpose to discuss technical surgical methods, and in fact the procedures used have been so varied, that a classification according to the type of operation would be a laborious undertaking. It is far more illuminating to divide the cases into two groups chronologically, the first period representing the pioneer work carried on in an attempt to develop a technically safe procedure, and the second period giving a fairer index of what may be expected today. As a medical consultant, I can only congratulate my surgical colleagues, Drs. Wyman Whittemore and Edward D. Churchill, that the years of disheartening and oftentimes tragic struggle with the operation of lobectomy seem at last to find adequate justification.

In the first ten cases (1921-27) nine total (right lower in five, left lower in four) and one subtotal (left upper) lobectomies, there were four operative deaths (40.0 per cent). In the next fourteen cases (1929-34) thirteen total (right lower in four, right middle in one, right lower and middle in three and left lower in five) and one subtotal (left lower) there was only one operative death (7.2 per cent). In the five remaining cases (1933-34) with only the first stage of the operation (separation of adhesions and the walling off of the pleural cavity preliminary to left lower lobe lobectomy) there has been one death. Thus, in the last nineteen cases there have thus far been only two operative deaths (10.5%).

Results of Operation in Surviving Cases. Not only has there been a striking drop in mortality, but the results of operation have improved.

Of the six survivors in the first group of ten cases (1921-27) one was completely relieved, three were much improved, one was unimproved, and one, improved, died three years after operation.

Of the thirteen surviving cases in the second group (1929-34) ten are entirely relieved of

cough and expectoration, but with a persistent bronchial fistula with a pin-point opening and occasional bleeding therefrom three years and nine months after operation in one, bronchial asthma, since as before operation, in one, and chronic empyema five months after operation in one. The remaining three with abundant foul sputum before operation still cough, one with one-half tablespoonful of non-foul sputum daily and the other two with about an ounce of non-foul sputum daily. In one the wound of operation is healed. In the other two a relatively short time has elapsed since operation and the persistence of a bronchial fistula to date is not significant.

Justification for Lobectomy. Though the chance of a fatality from lobectomy for bronchiectasis from the present operative procedures at the Massachusetts General Hospital cannot be definitely stated, it may be estimated as in the neighborhood of 10 per cent. There is a chance of incomplete relief from cough and expectoration from the presence of undiscovered lesions elsewhere than in the excised lobe and such postoperative complications as bronchial fistula and chronic empyema may occur in a small proportion of cases. The incapacity and suffering from the disease, risk of complications in untreated cases, failure of other methods, reduction in mortality from lobectomy and improvement in the postoperative results warrant resort to this operation in selected cases.

SUMMARY

Much progress has been made in the recognition of bronchiectasis during the past decade in consequence of advances in roentgenography and the use of lipiodol. The disease as an independent affection is more common than was formerly appreciated. The symptoms may suggest tuberculosis or lung abscess, but the nature of the disturbance can ordinarily be determined by adequate investigation. Conservative measures are disappointing in treatment. Collapse therapy may lead to improvement, but complete relief by this means is not to be expected. Lobectomy offers the prospect of cure. An encouraging reduction in mortality and improvement in the final results warrant resort to this operation in carefully selected cases.

STUDIES IN AGRANULOCYTOSIS. IV*

Report of Two Cases of Agranulocytosis Following the Use of Dinitrophenol

BY WILLIAM DAMESHEK, M.D.,† AND SAMUEL L. GARGILL, M.D.†

THE recent introduction of alpha-dinitrophenol for the treatment of obesity[1] has resulted in its enthusiastic acceptance by the medical profession and in its widespread use by the laity. Although the dangers attending the use of the drug have been recognized, they have apparently not acted as deterrents to its further use[2]. Recently a number of toxic and even fatal reactions have been reported, with hepatic or renal damage as the outstanding features[3].

Knowledge regarding agranulocytosis has recently been advanced by the reports of numerous observers[4] implicating amidopyrine and possibly other compounds as the probable etiological agents in about fifty cases. Kracke[5] had earlier enunciated the concept that the sudden rise to clinical eminence of this new syndrome might be due to the widespread use of drugs containing the benzene ring. In one report (Hoffman, Butt, and Hickey[1c]) the statement is made that dinitrophenol had been used by the patient prior to death and that it was probably of etiological importance.

The present report deals with two cases of agranulocytosis (one fatal) following the administration of alpha-dinitrophenol for the purpose of weight reduction. The first warning in both cases was given by involvement of the mucous membranes: the throat in one case, the gums in the second. No previous toxic reaction had occurred. When the blood was examined, profound leukopenia was found. Agranulocytosis was not considered in the first patient until she was in extremis and treatment with adenine sulphate was of no avail. In the second case, a leukocyte count was immediately made upon the appearance of gingival symptoms and treatment with adenine sulphate instituted within an hour. This patient recovered after an exceedingly stormy course.

Although it is not proved that the use of dinitrophenol bore anything but a temporal relationship to the development of agranulocytosis in the two cases, the definite possibility is present that the drug was of distinct etiological significance.

REPORT OF CASES

CASE 1. B. I. H. 21837.

M. G., an American Jewess, aged forty-six, was admitted to the hospital on March 26, 1934 and died the same day. In 1930 she had complained of a chronic urticaria which gradually cleared up. On February 9, 1934, her physician gave her alpha-

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dinitrophenol in a dosage of 200 mg. daily for the treatment of obesity. Later, the dose was increased to 300 mg. daily. Routine blood smears were normal, and she lost weight satisfactorily during the next month. On March 12, her physician found her to be in good condition; a blood smear showed no abnormalities. On March 23, she developed a sore throat and a slight elevation in temperature. The soreness failed to respond to ordinary measures. On March 25, the fauces were swollen and a grey necrotic area was present on the right tonsil. Dysphagia developed rapidly and the cervical lymph-nodes became swollen. On March 26, only three days after onset of illness, the patient was semi-stuporous, moderately cyanotic, and in extreme respiratory distress. On admission to the hospital, her temperature was 104.8° F., pulse 150, respirations 40. The leukocyte count, which was 1200 per cu. mm. on admission, fell to 400 and then to 100 per cu. mm. before death. No polymorphonuclear cells were present. One dose of adenine sulphate (1.0 Gm.) was administered intravenously. A tracheotomy was decided upon as an emergency measure and during this procedure the patient died. Permission for autopsy was not obtained. Questioning of the family indicated that she had not used amidopyrine or allonal. Her physician stated she had occasionally used phenobarbital.

CASE 2. B. I. H. 22493.

L. E., a single American Jewish bookkeeper, aged 40, was admitted to the Beth Israel Hospital on May 17, 1934. She had always considered herself well, but had gained 20 lbs. in weight during the past year, despite adherence to a low calorie diet. There was an unusual incidence of cardiovascular disease and diabetes in the family. Because she wished to lose weight, she consulted one of us on March 10, 1934 and expressed a desire for dinitrophenol, about which she had heard. Examination on that date was entirely negative except for moderate obesity. She was advised to continue her low-calorie diet but in addition was given capsules of alpha-dinitrophenol in 0.065 Gm. (1 grain) dosage, 2 capsules to be taken daily. She was seen at weekly intervals, and because no symptoms of toxicity had taken place, the amount of medication was increased to .195 Gm. (3 grains) daily and later to .325 Gm. (5 grains) daily. She lost 11 lbs. in weight during the period from March 10 to May 12 and felt unusually well. On the latter date, examination revealed no abnormalities. On May 16, because of pain in the gums, she consulted a dentist who told her she had Vincent's infection of the gums. That evening she developed a severe headache and felt feverish. On the following morning (May 17) she appeared extremely ill and semi-stuporous. There was a saffron-yellow tint to the skin, although the sclerae showed no icterus. The temperature was 104° F., the pulse rate 140. The gums were swollen and spongy and showed a flaky whitish exudate along the dental margins. The fauces showed diffuse redness with patches of whitish exudate on the right and on the posterior pharyngeal wall. Examination was otherwise negative.

It was thought the patient had dinitrophenol poisoning. Agranulocytosis was considered because of the lesions of the mouth and throat without other adequate cause for the marked elevation in temperature. White blood cell count was 875 per cu. mm., and the stained smear showed complete absence of polymorphonuclear cells. She was given 1.0 Gm. of adenine sulphate intravenously dissolved in 100 cc. of normal salt solution and was admitted to the hospital for further therapy.

On admission to the hospital, she was given 1000 cc. of normal salt solution together with glucose in 2.5 per cent concentration by hypodermoclysis. The urine showed a trace of albumin but was otherwise normal. Hemoglobin was 85 per cent, red blood cell count was 4.25 millions, and white blood cell count 400 per cu. mm. Another dose of 1.0 Gm. of adenine sulphate was administered intravenously.

On the following day (May 18), the lesions of the gums and throat were more extensive and showed areas of necrosis. Smears taken from the lesions failed to show the organisms of Vincent's infection. The leukocyte count was 600 per cu. mm. and no granulocytes were present, although the percentage of monocytes was elevated. A third dose of 1.0 Gm. of adenine sulphate was given intravenously together with 50 cc. of 50 per cent glucose. On May 19 she appeared worse. Temperature was 104° F., and the patient was drowsy and apathetic. There were extensive necrotic areas of the gums, mouth, and throat, and the teeth were loose. A fourth dose of adenine sulphate was administered. On May 20, temperature was 105° F., pulse 140. She was stuporous and semi-delirious. Leukocyte count was 700 and 800 per cu. mm. and one polymorphonuclear cell and one eosinophil were seen. She was given 2.0 Gm. of adenine sulphate intravenously together with 2000 cc. of normal salt solution and 2.5 per cent glucose by hypodermoclysis.

On May 21, for the first time, she looked better, although her temperature was still 104° F. The white blood cell count had risen to 1000 per cu. mm. and the differential count of the white cells showed 27 per cent granulocytes, most of them myelocytes and metamyelocytes. On May 22, there was an abrupt drop in temperature to 99.4° F., and the patient felt considerably better. The leukocyte count had risen to 5800 per cu. mm. and 42 per cent myelocytes were present among a total granulocyte count of 59 per cent. Convalescence from that time on was uneventful. The necrotic lesions of the mouth and throat healed rapidly with the rising leukocyte count, which reached its peak (14,700) on May 26. The patient has since been well.

DISCUSSION

Agranulocytosis appeared in both of the above cases after the constant daily use of dinitrophenol over a period of six weeks and nine weeks respectively in the recommended dosage of 300 mg. daily. Amidopyrine or mixtures containing amidopyrine had not been used. Phenobarbital had been occasionally used by the first patient. That the disease was caused by the drug because it appeared following its use is not subject to definite proof. However, the recent developments in defining the etiology of agranulocytosis, the close relationship of the drug to benzene in chemical structure, its profound effects on cellular metabolism, its well-known toxicity to hepatic and renal cells all lead one to suspect it strongly in these cases, especially since no other etiological agents were readily discernible.

Conceptions regarding agranulocytosis have shifted considerably since the first reports were published. The blood-picture was at first considered to be secondary to a severe infectious process, usually of the throat. The observation that many cases were ushered in by a rapidly

developing leukopenia which was followed by necrotic lesions of various mucous membranes indicated that the infection was often secondary to the leukopenia rather than the primary factor[6]. The cause of this "selective" aplasia of the bone-marrow resulting in disappearance of the polymorphonuclear cells from the circulating blood was not known, although metabolic, constitutional, and hormonal factors were variously suspected. Kracke in 1931[5] attempted to incriminate benzol and the benzene ring which was prevalent in so many widely-used pharmaceutical compounds. He advised in 1932[7] that physicians "should obtain thorough and complete histories with especial reference to the use of unusual foods, drugs, cosmetics, clothing, shoe and hair dyes, and other substances of a rare nature that may contain bone-marrow depressing substances, as benzene or its allied products". Among the first definite reports incriminating a drug other than arsphenamine was that of Madison and Squier[4b] who found that of fourteen patients developing agranulocytosis, thirteen had taken amidopyrine either alone or in combination with other drugs. Numerous reports followed, substantiating these observations and incriminating amidopyrine or mixtures containing this compound[4]. The ordinary barbituric acid compounds and acetylsalicytic acid did not appear to have etiological significance. Kracke[8] later concluded that it was the "benzamine" drugs (containing the benzene ring together with the attached NH_2 or NO_2 grouping) which were the outstanding etiological agents.

In view of these observations, it is not surprising that a substance with such profoundly metabolic effects as dinitrophenol should now be suspected of causing the disease. This chemical containing the benzene ring and closely related to the explosive trinitrotoluol ("T. N. T.") in composition ($C_6H_3OH\ (NO_2)_2$) apparently has its chief action on muscular and other bodily cells, greatly increasing their oxidative processes without, curiously enough, causing an increase in the pulse rate or the velocity of blood flow[9].

In the rapidly increasing literature on the toxic effects of dinitrophenol, there is surprisingly little mention of its effects on the bone-marrow. Hoffman, Butt, and Hickey[4c] in their series of cases of agranulocytosis following the use of amidopyrine reported one case following the administration of dinitrophenol for the purpose of weight reduction.* Despite the close relationship of the drug to benzol, neither anemia nor purpura has yet been described. The outstanding toxic effects that have been mentioned are hyperthermia with profuse sweating, urticaria, angioneurotic edema, jaundice, and renal damage[10]. Masserman and Goldsmith[11] observed tachycardia, marked reduction in blood-pressure, acidosis and progressive stupor in five cases, one of which terminated fatally.

*One of us was told by Dr. R. R. Kracke at the 1934 A. M. A. Convention in Cleveland that in four days (June 11-15, 1934) he had been informed by various physicians of sixteen cases of agranulocytosis, most of them fatal, which occurred during treatment with dinitrophenol.

The mechanism attending the toxic effects of the drug has not been definitely established. The occurrence of urticaria and angioneurotic edema in certain individuals, and the marked individual variability in tolerance of the drug both suggest an allergic nature of certain of the reactions. Mayer[12], in his studies of the effects of various chemicals on munitions workers, found wide variations in susceptibility. We have found the same in the treatment of a group of patients with obesity.

That the drug dinitrophenol is a treacherous one has already been pointed out by Poole and Haining[13], who state that it appears to induce a relative euphoria or stimulation due to hypermetabolism when given in subtoxic doses. Another even more treacherous property is that seen in the two cases described above, both of which developed agranulocytosis without previous symptomatology and only a few days after careful medical supervision.

In view of the striking differences in individual susceptibility to the drug, its unpredictable toxic properties, and its rapidity of action in inducing agranulocytosis, at least as seen in our two cases, it is felt that the dangers of dinitrophenol far outweigh any therapeutic advantages which might be obtained. Moreover, it has been our experience that the drug must frequently be given in maximal therapeutic dosage to obtain even a slight loss in weight.

The use of adenine sulphate given intravenously in the second case appeared to be a life-saving measure. This conforms with our experience with nucleic acid derivatives in six other cases[14]. Of nine cases treated without nucleic acid derivatives, seven died. Of seven recent cases treated intensively with either a mixture of pentose nucleotides or adenine sulphate, all are alive and well. Adenine sulphate given intravenously appears to act more rapidly and to cause fewer reactions than "pentnucleotide" which is given intramuscularly. In one patient who failed to respond to pentnucleotide, recovery ensued following the use of adenine sulphate.

SUMMARY

Report is made of two patients who developed severe agranulocytosis while taking dinitrophenol in ordinary therapeutic dosage for the treatment of obesity. One patient died, the condition of agranulocytosis not being recognized until the day before death. Diagnosis in the second patient was made immediately upon the appearance of gingivitis, and treatment with

adenine sulphate was successfully carried out despite an exceedingly stormy course.

Despite absence of definite proof, it seems logical to conclude that the agranulocytosis in these two cases was caused by the drug dinitrophenol. Attention is called to the various toxic reactions of this widely-heralded drug and the dangers which might attend its continued use as a therapeutic agent. The concept that agranulocytosis may be precipitated in certain instances by drugs containing the benzene ring in association with the NH_2 or NO_2 grouping appears to be borne out in these cases.

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PURPURA HEMORRHAGICA WITH PROFUSE BLEEDING FROM THE MUCOUS MEMBRANES FOLLOWING THE TREATMENT OF SYPHILIS WITH BISMARSEN*

BY J. L. GRUND, M.D.†

ONE of the relatively newer antisyphilitic drugs used to a greater or lesser extent in the treatment of syphilis at present, is bismarsen. Synthesized by Raiziss in 1924, it represents the result of an attempt to combine in one molecule two of the most widely used and effective spirocheticidal elements, arsenic and bismuth. It is the sodium salt of a bismuth derivative of arsphenamine methylene sulphonic acid with an arsenic content of from 13 to 15 per cent and a bismuth content of from 23 to 25 per cent.

Raiziss and Severac[1] have reported results which indicate that bismarsen is as effective as arsphenamine in experimental rabbit syphilis. Numerous clinical studies have been made with the compound, the most extensive of which have been those of Stokes and Chambers[2], O'Leary[3], Stokes, Miller and Beerman[4] and Kolmer[5]. A review of the results obtained by the preceding points to the following. The spirillicidal and healing effect of bismarsen is somewhat slower than that of other arsphenamines; its ultimate effect in early syphilis, however, is equal to that of combined treatment with other drugs (Stokes and Chambers[2]). It was observed by O'Leary and Montgomery[6] to be satisfactory in acute syphilis in the elderly and in the serologically negative stage of chancre. Kolmer obtained good results in congenital syphilis, in pulmonary spirochetosis and in severe cases of Vincent's angina. We have used bismarsen since its introduction and have noted also that it may be used with good results in congenital syphilis (in which stage, however, it is not superior to intramuscular sulpharsphenamine) and in cases in which the veins are absolutely inaccessible.

As with other arsphenamines, for bismarsen must essentially be considered an arsphenamine, untoward reactions may occur with its use. Stokes and Chambers noted reactions but called attention to their lesser frequency and milder character. As regards the untoward reaction of arsenical dermatitis, it was Kolmer's experience that those cases which manifested this unwanted by-effect with intravenous arsphenamine presented similar cutaneous changes when bismarsen was substituted. At our clinic, in which bismarsen has been used since its appearance, cases intolerant to the arsphenamines presented exactly similar symptoms of intolerance when given bismarsen. Patients, in whom backache or nausea and vomiting were evident either with or shortly after an intravenous administration of an antisyphilitic arsenical, suffered similar symptoms after treatment with bismarsen, with the added feature that instead of appearing with

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or shortly after treatment, they appeared a number of hours later, a circumstance which prevented intelligent treatment of the patient's symptoms since he had already left the clinic. The matter of time in these cases is explained by the slower absorption of bismarsen from its intramuscular site of injection. One private case who presented a beginning dermatitis following intravenous neoarsphenamine, was given intramuscular bismarsen following the subsidence of his cutaneous lesions and responded with a diffuse urticarial eruption five hours after his treatment. It was also apparent that although untoward symptoms were of less severity following bismarsen than when intravenous therapy was pursued, they were of an essentially similar character indicating the presence of either an inherent intolerance or an acquired hypersensitivity to the arsenobenzol radical in both the arsphenamines and the bismarsen.

The great care which must be exercised as regards the future antisyphilitic treatment of patients who have suffered an arsenical dermatitis and have recovered has been emphasized by C. Morton Smith. More recently Fasal[7] in studying 125 cases with skin lesions following salvarsan treatment at the Vienna Dermatologic Clinic in the past five years called attention to the necessity of omitting future arsenical treatment in these patients. Practically all of his patients who manifested a salvarsan dermatitis and had recovered presented a recurrence of skin lesions when arsenical treatment was re-instituted. This fact must be kept in mind when substitution of the intravenous arsphenamines with some other form of antisyphilitic therapy is called for. And especially must care be exercised in the choosing of the drug to be used because of the fatal outcome of the occasional case of arsphenamine dermatitis.

It has been noted that since the introduction of bismarsen the tendency has been to resort to it when difficulties arise which militate against the carrying out of accepted forms of antisyphilitic treatment. The facile manner in which bismarsen is administered, namely by means of the intramuscular route, has engendered so great a sense of security with its use, that it is given by many routinely in all cases and in all forms of syphilis in preference to the long-established and accepted arsphenamines. Sight is evidently lost of the fact that bismarsen is a sulphoxylate arsphenamine and that like sulpharsphenamine which has been entirely discarded in adult syphilis it is capable of causing injury to the hematopoetic system. It is with the preceding in view that the following case is reported.

A. L., male, aged forty-eight, was referred to the dermatologic clinic of the Massachusetts Memorial Hospitals in October, 1928 because of a positive blood Wassermann test. Physical examination showed a gumma over the left knee.

From his entrance to the clinic until July, 1930, he received a total of twenty-five intravenous injections of neoarsphenamine and forty-five intramuscular injections of bismuth. Although his syphilitic gumma entirely disappeared, we were unable with this treatment to effect a favorable change in his serology. There was no evidence of signs of intolerance to the treatment at any time.

Inasmuch as the patient showed no other signs of syphilis than a persistently positive Wassermann, he was considered Wassermann-fast. At the time, we were carrying out an attempt to verify Stokes' "successful" use of bismarsen in Wassermann-fast syphilis and placed the patient under treatment with the compound[8]. From August to December, 1930, he received a total of twenty intramuscular injections of bismarsen without any change in the blood. Further treatment with neoarsphenamine up to December, 1930, was similarly without effect. No untoward reaction was apparent at any time during or after treatment. All forms of treatment were evidently well tolerated.

He was given no further antisyphilitic medication until May 16, 1931, when, following an injection of 0.45 Gm. of neoarsphenamine, after an interval of eleven hours, many purplish blotches appeared on his legs. The next morning his entire body was covered with very many reddish blue spots. He vomited and spit blood, his urine was bloody and there was bleeding from his rectum. He came to the Out-Patient Skin Clinic and was referred to the house service. Blood studies here showed normal red and white and platelet counts, a bleeding time of ten minutes and a clotting time of three minutes. After a three days' stay in the hospital, he was discharged as well.

He returned on August 3, 1931, and was given an intramuscular injection of 0.2 Gm. bismarsen. He was seen at the clinic two days later and presented over his entire body, numerous areas of petechial hemorrhage. He stated that they appeared in the evening of the day he received the bismarsen. Areas of hemorrhage were evident on the mucous membranes of his mouth and tongue. Bleeding from his nose, gastro-intestinal and urinary systems were also noticed. He refused to enter the hospital and was not seen until August 15, 1931. Blood studies previous to this time were therefore not done. On August 15, however, his hemoglobin was 75 per cent, his red blood corpuscles 3,790,000 and his platelets 330,000. The white blood count was normal. The bleeding time was one and one-half minutes and the clotting time one minute. He presented no signs or symptoms of his blood disturbance.

COMMENT AND CONCLUSIONS

In considering the present case, a brief review of the various blood disturbances that may occur with the use of the arsphenamines is necessary. One of the best and most comprehensive works dealing with this subject is that of McCarthy and Wilson[9]. They have divided the blood dyscrasias following the arsphenamines into three categories. The first has been classed as the thrombocytopenic. Recovery in this group is the invariable outcome and purpura and external hemorrhages are the presenting symptoms. The latter occur anywhere from several hours to five days after treatment with the

arsenical. There is no evidence of depression of the bone marrow function. Anemia when present is explained by the loss of blood as a result of the hemorrhages. The only pronounced change that may be present is a lowering of the platelet count directly attributable to a destruction of the circulating blood platelets. This is considered a peripheral phenomenon of, in all probability, an anaphylactoid nature and develops as a rule after a considerable number of injections. A state of sensitivity, specific to the drug used, is established. This could very well explain the phenomenon of the peripheral destruction of blood platelets, transient in character, occurring with the administration of the drug and passing relatively rapidly with recovery, after cessation of the offending treatment. Estimation of the degree of the lowering of the platelet count in this particular type can be ascertained only if it is carried out either with or immediately following the onset of the symptoms.

The second type of blood reaction is the granulocytopenic. In this subdivision, the mortality is 33 per cent. Recovery occurs if agranulocytosis is absent. Fever and angina may be present. Moderate anemia is the rule; the white cell count is low but there is no diminution of blood platelets.

The third type is the aplastic. The mortality in this group is very high, 83 per cent. Should recovery occur, the convalescence is very slow. The red cell count is usually reduced to below two million. A pronounced leukopenia is present and the platelets are low in number. Epistaxis, purpura and bleeding from the mucous membranes are evident, which together with the preceding blood picture are strongly indicative of a profound depression of the bone marrow.

The present case though manifesting several symptoms belonging to the third group, namely epistaxis and profound purpura with marked bleeding from the mucous membranes, must be classed as a thrombocytopenic bismarsen dyscrasia. The practically normal red and white blood and blood platelet counts eliminate the last two mentioned blood dyscrasias from consideration.

Occasional comments have appeared of such a nature as to question just which portion of the bismarsen is at fault when untoward reactions are encountered with its use. The fact that in the case described, in addition to the nature of the reactions noted in others of our cases treated with bismarsen, a certain train of symptoms was evident following neoarsphenamine, to be followed by an exactly similar course of untoward symptoms when bismarsen was substituted, seems to prove that the arsenic component is the etiologic factor. In connection with the latter, Wise and Sulzberger[10] are of the opinion that, in these cases, there appears to be an idiosyncratic hypersensitivity to arsphenamine, and that the hematopoietic system becomes sensitized to this compound and acts as the shock tissue with the result that repeated shock reactions in the blood building organs, which in the process have become specifically sensitized to a variety of circulating allergens, are responsible for the appearance of a blood dyscrasia. In this instance the sulphoxylate arsphenamine portion must have been the offending allergen.

The case described further demonstrates that bismarsen cannot be used entirely without compunction and that the same care and caution must be exercised with its employment as must be practiced with the other arsphenamines. Such an essentially serious untoward reaction as dermatitis or blood organ destruction may with it, as with the other arsphenamines, be a dangerous complication.

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STUDIES IN TUBERCULIN REACTIONS

The Scarification, Multiple and Single Puncture Technique

BY ELI FRIEDMAN, M.D.,* AND JOHN B. HAWES, 2ND, M.D.*

SINCE 1907, when von Pirquet[1] first introduced it, the use of the cutaneous tuberculin test has become universal. The technique as usually carried out is briefly as follows: The skin of the flexor surface of the forearm is thoroughly cleaned and dried. A drop of undiluted old tuberculin is then placed on the skin and an area of epidermis four to six mm. in diameter is removed by a twisting motion through the tuberculin with a small sterilized drill or Pirquet borer made for this purpose. This scarification is supposed to reach and expose the vascular cutis. After a few minutes, the excess tuberculin is carefully wiped off. A control is made on the same arm a few inches away, using sterile salt solution instead of tuberculin in order to determine the amount of reaction resulting from trauma alone. The scarification is inspected at the end of twenty-four and forty-eight hours. Without going into a detailed description of the various types of reactions, most observers consider an elevated papule with an area of erythema more than five millimeters in diameter at the end of the twenty-four and forty-eight hours as a positive reaction.

In 1916, Craig[2] introduced the multiple puncture or tattoo method of performing this test. His method corresponds to Hill's[3] multiple puncture technic in vaccination against smallpox. The arm is prepared as for the Pirquet test. An ordinary sewing needle is used. The needle, which should be new, sharp, and sterile, is held with the forefinger and middle finger above, and the thumb below, with the point of the needle at right angles to the patient's arm. Held in this way, nearly parallel to the skin, the side of the needle is pressed firmly seven to ten times into the skin through a drop of tuberculin in an area of about ½ cm. in diameter. The point of the needle with this technic is not driven directly into the skin. Each time pressure is applied at right angles to the shaft of the needle, the elasticity of the skin will pull a fraction of an inch of the epidermis over the point of the needle, so that the tuberculin is carried into the deeper layers of the epidermis. No sign of bleeding should occur and all evidence of trauma should disappear in a few hours. The epidermis is left practically intact and in the vast majority of cases there is no evidence of trauma in the control.

In 1927 Chester A. Stewart[4] introduced the single puncture technic for performing the von Pirquet test. In this method, the arm is prepared in the usual way, a drop of undiluted tuberculin is placed on the forearm, and an ordinary sewing needle is introduced, with a single puncture, through the tuberculin into the dermal layers of the skin for a distance of about one-quarter of an inch. The excess tuberculin is immediately wiped off, and as usual, the reaction is read at the end of twenty-four and forty-eight hours.

In this study, in order to avoid a possible constitutional reaction from absorption of too much tuberculin, we did not do all the tuberculin tests simultaneously, but first tried to determine the comparative merits of the multiple puncture and scarification technique, and then carried out a similar study on a different group of children to determine the relative merits of the multiple but comparatively superficial punctures with the single but deeper puncture. Our first step, therefore, was to compare the Craig or multiple puncture method with the old time scarification with a blunt instrument, or Pirquet borer. Our conclusions are based on the results of the above tests done on 100 children at the Prendergast Preventorium of the Boston Tuberculosis Association. Each child was a contact case and each had previously reacted positively to the von Pirquet test before admission. Both tests were done on the same arm because it has been shown that in certain individuals the reactivity of the skin in different parts of the body may vary considerably. A Mantoux or intradermal test was also done on the same arm in the dilution of 1 to 10,000 for the purpose of comparison. The dilution of 1 to 10,000 was chosen instead of the ordinary dilution of 1 to 1,000 used in the regular Mantoux test because, according to Happ and Casparis[5], a von Pirquet test introduces only .01 mg. of tuberculin into the skin. We used human tuberculin provided by the Massachusetts State Department of Health.

To prevent the merging of the three reactions which in extremely sensitive individuals might occur, it was found advisable to arrange the scarification, intradermal and multiple puncture tests in triangular fashion on the forearm. These reactions were read in twenty-four, forty-eight and seventy-two hours respectively, and, depending on the amount of erythema and induration, they were classified as slight, moderate, or severe. Controls with sterile salt solution were also done on the opposite arm using the scarification and multiple puncture technic. No

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attempt was made to correlate the severity of the tuberculin reaction with the degree of activity of the tuberculous infection.

The following tables present the details of our observations on the different tests.

TABLE I

TYPES OF REACTIONS IN 100 CHILDREN WITH THE INTRADERMAL, SCARIFICATION AND MULTIPLE PUNCTURE TESTS

Tests	Severe	Moderate	Slight	Negative
Intradermal	7	43	50	
Scarification	7	37	41	15
Multiple puncture	7	44	49	

Any reaction showing an area of erythema of ½ cm. in diameter, especially if associated with a certain amount of induration, was considered positive. Those reactions that did not differ from the controls were classified as negative. All types of reactions were noticed in these cases. The majority appeared within the first twenty-four hours as a red elevated papule varying from one half to four cm. in diameter and lasted for several days, fading gradually on the fifth day. Some of the severe reactions showed vesiculation, marked erythema and induration, while a few showed lymphangitis radiating from the site of test. All these severe reactions lasted for several weeks.

In fifteen of this series, the scarification tests were negative. Four were classified as negative because the reactions did not differ from the controls, while eleven were negative probably because of too superficial scarification since they were positive when repeated with the proper technic. It is interesting that of the scarification tests which were negative because of too superficial scarification, eight were found in twenty children in whom the tests were done by a nurse inexperienced in this work. It is likewise of interest that the multiple puncture tests done by this same inexperienced worker on the same twenty children were all positive.

TABLE II

AVERAGE SIZE OF LOCAL REACTIONS IN 24-48-72 HOURS IN THE THREE TESTS

Tests	24 hours	48 hours	72 hours
Intradermal	1.4x1.4 cm.	1.5x1.4 cm.	.9x1 cm.
Scarification	.9x.9	.9x.9	.5x.6
Multiple puncture	1x1	.9x.9	.6x.6

A few reactions were of the "premature" type characterized by rapid evolution, reaching the maximum in ten hours and disappearing on the second day. There was only one case that seemed doubtful after twenty-four hours and definitely positive after forty-eight hours. It would seem advisable therefore, to examine tests in twenty-four hours so as not to miss the "premature" type of reaction and also in forty-eight hours if a reaction seems doubtful on twenty-four hour examination.

TABLE III

FREQUENCY AND SIZE OF LOCAL REACTIONS IN CONTROLS

Tests	Reactions Present	Average Diameter cm.
Scarification	100 per cent	.5
Multiple puncture	25 per cent	.3

From the above table, it is evident that the scarification technique almost always leaves a traumatic area of about one-half centimeter in diameter, while the multiple puncture test leaves no traumatic reaction in the vast majority of cases and only slight reaction in a few. In severe and moderate reactions, the interpretation is comparatively simple, as there is definite vesiculation and induration with the erythema, but when the reaction is mild, it is often difficult to tell whether the erythema is due to trauma, sensitiveness to tuberculin or nonspecific sensitiveness to other substances found in the culture media such as glycerine, extractive of beef bouillon, salts, etc. For this reason, some observers do not call a tuberculin test positive unless there is an area of erythema of 1 cm. rather than ½ cm. in diameter.

It is obvious from our observations that the multiple puncture or "tattoo" technic for the cutaneous tuberculin test has many advantages over the orthodox scarification method of von Pirquet. It is distinctly less painful. With the needle held almost parallel to the surface of the skin the sharp point of the needle penetrates the epidermis sufficiently to give us by this method, as Craig suggested, a combination of the cutaneous and intracutaneous tests, without causing much pain. Practically all the children agreed that the multiple puncture method was the least painful.

Another advantage of the multiple puncture method is the greater certainty of the introduction of tuberculin into the deeper layers of the skin by at least a few of the many punctures made with a sharp-pointed needle, as compared with the scarification method especially when done with a blunt instrument. Dienes and Mallory[6], in their study of hypersensitive reactions, emphasize the fact that tuberculin sensitivity is due to antibodies fixed upon the cells, and that there is also a tendency to selective epithelial necrosis not noted in the anaphylactic type of reaction, where circulating antibodies are chiefly responsible for the reaction. This explains why it is essential, for a successful tuberculin test, to bring the tuber-

culin into intimate contact with the tissue cells. With the scarification method even an experienced worker often finds it difficult to penetrate to the desired depth on account of the relative dullness of the "borer" and of the variability of the texture and thickness of different skins. This source of error has been demonstrated definitely in our series, where eight tests out of twenty were negative the first time and positive the second time, when done properly. This observation conforms with the experience of numerous other investigators who found a larger percentage of reactors to the von Pirquet test upon repetition. Nothmann[7], for instance, found that out of 263 children 47 per cent reacted to the first von Pirquet and 65 per cent reacted when the test was repeated, while Bass[8] noted that 3.3 per cent of the children tested reacted to the first von Pirquet test done by the scarification method, 6.8 per cent reacted to the second and 10 per cent reacted when the test was repeated for the third time. That this wide variation in the percentage of reactors to the first and to subsequent tuberculin tests is due, in part at least, to variations in technique, rather than to the sensitizing influence of previous tuberculin inoculations is apparent from the fact that other observers such as Heimbeck[9], and Aronson, Zacks and Poutas[10], using strict and uniform technique, found in their series that only 0.8 per cent more reacted to the second von Pirquet than to the first.

Our next step was to determine the relative merits of the multiple but comparatively superficial puncture method with the single but deeper puncture. This study was carried out on a new group of seventy-five children at the Prendergast Preventorium. These children, as in the last group, were all definite contact cases who have at some previous time shown a positive reaction to tuberculin. As a standard for comparison an intradermal test of the same dilution of 1 to 10,000 used in the first group was given simultaneously with the multiple and single puncture tests on the same forearm. Controls of the single and multiple punctures were done on the opposite arm with salt solution. Reactions were read at the end of 24, 48, and 72 hours.

TABLE IV

AVERAGE SIZE OF LOCAL REACTIONS IN 24-48-72 HOURS IN THE THREE TESTS

Tests	24 hours	48 hours	72 hours
Intradermal	1.5x1.6	1.6x1.7	1x1
Multiple puncture	1.1x1.1	1 x1	.7x.7
Single puncture	1.2x1.2	1 x1	.8x.8

Comparing the single puncture method with the multiple puncture method we find that they agree in 100 per cent of the cases. In no instance was one positive and the other negative. Although the individual reactions were a trifle more papular and pronounced in the single puncture technic, the area of erythema corresponded very closely. The fact that the single deep puncture was a little more painful was more than compensated by the speed with which this test was done as compared with the superficial but multiple punctures. There was no constitutional reaction noted in any of the children of either group.

From our observations it is therefore fair to state that the single puncture method is fully as reliable as the multiple and in some ways preferable because of the speed with which this test can be done. Emanuel Friedman, Merwin H. Black, and A. L. Esserman[11] in their study of a group of fifty-five children state that they obtained 7 per cent fewer reactions with the multiple than with the single puncture technique and make the rather extravagant claim that the single puncture method yields results as accurate as those obtained by the well-established Mantoux test. We feel that this statement is unwarranted without more extensive investigation.

TABLE V

FREQUENCY AND SIZE OF LOCAL REACTIONS OF CONTROLS

Tests	Frequency of Reactions	Average diameter cm.
Multiple puncture	28 per cent	.4
Single puncture	20 per cent	.3

From the above table it is very evident that the local traumatic reactions found so objectionable in the scarification method with a blunt instrument are even less marked in the single than in the multiple puncture method.

SUMMARY AND CONCLUSIONS

A study of the comparative merits of the scarification, single puncture, and multiple puncture methods, on two different groups of children at the Prendergast Preventorium, leads us to conclude that both the single and multiple puncture methods are more satisfactory than the old orthodox scarification method for the following reasons:

1. Both the single and multiple puncture methods are less painful.
2. The technique is simpler.
3. No special instruments are necessary, as an ordinary sewing needle is always available.
4. The incidence of too superficial scarification is entirely eliminated, since the tuberculin is carried into the deeper layers of the skin by both the single and the multiple puncture methods.

5. There is less traumatic reaction because it is not necessary to remove an area of epidermis to bring the tuberculin in contact with the deeper layers of the skin as in the scarification method, hence the interpretation of mild reactions is greatly simplified.

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SPONTANEOUS MENINGEAL HEMORRHAGE*

Case Report

BY R. CANNON ELEY, M.D.†

THE term idiopathic or spontaneous meningeal hemorrhage has been used to describe a symptom complex characterized by the sudden onset of signs of meningeal irritation and the finding of bloody or xanthochromic cerebrospinal fluid by lumbar puncture, together with prolongation of the bleeding and coagulation times of the blood, and the formation of a non-retractile, easily re-dissolved blood clot. The condition, spontaneous meningeal hemorrhage, was described as a definite clinical entity by Ehrenberg[1], but very little has been published about it since that time. The French and German writers have also called attention to this disorder, but a surprisingly small amount of interest has been given to it. In Neal's[2] group of thirty cases the most outstanding symptom was headache, which was sudden in onset and severe in its intensity.

The pathogenesis of spontaneous meningeal hemorrhage has not been satisfactorily explained. Symonds[3] and Fearnsides[4] suggest that the cause of the hemorrhage may always be found in a degenerative process of the arterial wall which may result from any acute or chronic infection. In patients who are too young for such a process to have occurred, the suggestion is made that there is a congenital defect in the media of the vessel wall which is conducive to the development of aneurysms which may subsequently rupture. Postmortem examinations do not support this view. Goldflam[5] states that the disease is due to a true diapedesis dependent on some dysfunction of the vasomotor control and probably of venous origin. Weil, Isch-Wall and Pollet[6] have demonstrated changes in the blood comparable to those seen in hemorrhagic conditions, i.e., prolonged bleeding and coagulation time with the formation of a poorly retractile clot. In some of Neal's cases other diseases which were present may have exerted some influence in the production of the meningeal hemorrhage. However, she does not place much importance on the presence of these diseases and is of the opinion that they are of no etiological importance[7]. When one takes into consideration the prolonged bleeding and coagulation time, the non-retractile and easily re-dissolved blood clot, and the slightly reduced number of blood platelets it would appear that the condition is very closely related to some dyscrasia of the blood per se rather than the result of anatomical or histological changes within the walls of the blood vessels. However, a combination of these two conditions, i.e., a hemorrhagic tendency of the blood, and histologically imperfect blood vessels, might easily result in the production of this clinical entity as both factors have been described in these patients. Obviously, further studies must be made before any final conclusions can be reached in regard to the etiology of this disease.

The diagnosis can usually be made without great difficulty although at first the signs and symptoms may so simulate other conditions that some confusion may exist before the correct diagnosis can be established. Such conditions as trauma, subdural hematoma, meningitis, hemophilia and purpura hemorrhagica have to be considered. As a rule trauma can be excluded by a carefully obtained history and physical examination; hematological studies do not support a diagnosis of subdural hematoma; the absence of organisms within the cerebrospinal fluid as demonstrated both by direct smear and by bacteriological cultures, and the clinical course differentiate this disease and meningitis; the past history of the patient, the fact that the

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condition occurs in both males and females, and the prolonged bleeding time as well as coagulation time preclude the diagnosis of hemophilia. Longcope[8] has reported a series of patients who had cerebral and spinal manifestations associated with purpura hemorrhagica and it is quite possible that spontaneous meningeal hemorrhage might at first be confused with this syndrome. However, in the former disease the coagulation time is normal and the platelets are reduced to a lower level than has been observed in the reported cases of spontaneous meningeal hemorrhage. Buckman[9] has described a group of five children who suffered from prolonged bleeding following injuries, all of whom had prolonged coagulation time and bleeding time. The blood platelets were normal or increased in number. This observation distinguishes the condition from idiopathic or spontaneous meningeal hemorrhage. In brief, the special features characteristic of this clinical entity are prolonged bleeding and coagulation times of the blood with the formation of a poorly retractile, easily redissolved friable blood clot; the uniform distribution of blood in successive specimens of the cerebrospinal fluid; the failure of this blood to form a clot when allowed to stand, and the presence of supernatant xanthochromic fluid when a specimen of the cerebrospinal fluid is centrifugalized.

In view of the prolonged bleeding and coagulation time and the reduced number of blood platelets, such measures as will tend to restore these to within normal limits are indicated, and constitute the chief mode of treatment. Of the various measures that may be employed in an effort to control these factors, a transfusion of either whole blood or citrated blood has proved to be most effective. Increased intracranial pressure can be temporarily relieved by lumbar puncture with removal of the cerebrospinal fluid, although care must be exercised so as to avoid respiratory complications resulting from rapid lowering of the cerebrospinal fluid pressure. Absolute quiet and rest in bed are just as important in the treatment of this condition as they are in any case of intracranial injury.

The prognosis is usually good, although recurrences are not of favorable import and may cause a fatal termination. Of the cases reported by Neal, twenty-one of the thirty-five recovered, and in those that died the cause could often be attributed to some other existing condition. The onset of illness is usually the height of the disease and as the bleeding subsides the patient usually shows a progressive improvement, and makes a gradual and uncomplicated recovery.

As the disease is not a common condition and is encountered rather infrequently the following case report is of interest.

CASE REPORT: J. C., a white male child, 11 and 1/3 years of age, was admitted to the hospital on account of violent headache, vomiting, and a stiff neck. The family history and past history were unimportant and there was no history of trauma. The present illness began twenty-eight hours prior to admission when the patient suddenly complained of a severe headache. Shortly afterwards he vomited. For the following twelve hours he remained drowsy and slept quietly, but on awakening, vomiting and headache recurred and his neck became stiff. He was then examined by his family physician and in view of the signs and symptoms suggestive of meningeal irritation a lumbar puncture was performed under ether anesthesia and grossly bloody cerebrospinal fluid was obtained. However, as it was thought that the patient was suffering from some type of meningitis he was referred to the hospital for further study.

Physical examination revealed a well-nourished and normally developed white male child resting fairly comfortably in bed but holding his head between his hands, and complaining of severe headache. His face was flushed, respirations were regular and even, and the sensorium was intact. T. 99.6; P. 30; R. 20. Blood pressure 105/60 mm. of mercury. The skin was moist, elastic and free from rashes or petechiae. Percussion of the skull did not reveal a positive Macewen or cracked-pot sound and there were no gross abnormalities. The ears, nose and throat were normal. Extraocular movements were normal, the pupils equal and reactive to light, and the fundi did not present any evidence of increased intracranial pressure. The neck was stiff, retracted, and resisted anterior flexion. The heart and lungs were normal to percussion and auscultation. The abdomen was soft, flat, and free from areas of tenderness or palpable viscera. The genitalia and extremities were normal. The spine was held in a rigid manner and there was slight opisthotonos. The deep reflexes were exaggerated and there was a bilateral Kernig sign. Examination of the blood showed hemoglobin 90 per cent; red blood cells 5,000,000 per cu. mm.; and white blood cells 18,000 per cu. mm. with 83 per cent polymorphonuclear leucocytes. The Wassermann and tuberculin tests and urinalysis were negative.

In view of the clinical manifestations a tentative diagnosis of meningitis was made and a diagnostic lumbar puncture carefully performed under local anesthesia. The cerebrospinal fluid removed at this time was grossly bloody but as a similar fluid had been obtained by lumbar puncture just a few hours prior to the patient's admission to the hospital it was believed that this finding of blood could not be explained by trauma. Before the needle was withdrawn from the lumbar subarachnoid space 10 cc. of antimeningococcus serum were administered. Direct smears of the cerebrospinal fluid failed to show any organisms, and cultures were negative for organisms. On the morning after admission the patient appeared clinically improved although he still presented evidence of meningeal irritation, and complained of severe headache. A second lumbar puncture was performed and fluid similar to that obtained on the previous puncture removed. Antimeningococcus serum was again administered intraspinously. As no organisms could be demonstrated in the second specimen of cerebrospinal fluid, and as all bacteriological cultures were negative, it was thought advisable to observe the patient carefully for the next few hours and not to repeat the lumbar punctures unless evidence of increased intracranial pressure appeared.

It was observed that the cerebrospinal fluid presented the same degree of blood tinging in several successive tubes and that the blood in the fluid failed to coagulate. Furthermore, when a specimen

of the fluid was centrifugalized the supernatant portion was xanthochromic. (In traumatic lumbar punctures the amount of blood in the cerebrospinal fluid varies, being greatest in the first fluid withdrawn, and least in the last fluid removed. On standing the blood readily clots and a centrifugalized specimen does not show xanthochromic fluid.) These observations immediately suggested the probability of an intracranial hemorrhage, either secondary to an acute infection or to some blood dyscrasia. As bacteriological examinations of the cerebrospinal fluid had consistently been negative, and as no organisms had been obtained on repeated cultures of the blood, infection could be satisfactorily excluded. Hematological studies gave the following information; bleeding time ten minutes; coagulation time eight minutes with a friable, non-retractile clot which readily redissolved on agitation; fragility test normal; reticulocytes normal and platelets 150,000 per cu. mm.

In view of these observations, i.e., prolonged bleeding and coagulation time, somewhat reduced platelets, and friable blood clot, the patient was transfused with 225 cc. of citrated blood. This procedure was followed by a decided improvement. The bleeding time was reduced from ten minutes to three and a half minutes, and the clot was firm and retractile. The cerebrospinal fluid, removed by lumbar punctures made at repeated intervals during the patient's uneventful convalescence, showed a gradual transition from a xanthochromic color to the water-clear color of normal cerebrospinal fluid. Fourteen days after admission the patient was discharged from the hospital.

During the following six months the child enjoyed good health and led a normal life in all respects. At this time he suddenly developed a severe headache, his neck became stiff and he was readmitted to the hospital. There was no history of trauma or of any preceding illness. The physical examination presented similar findings to those described at the time of his former admission with the exception of the pulse which was weak, slow (rate 40-50 per minute), irregular and could not be felt at the wrist. A lumbar puncture was immediately performed and blood-tinged cerebrospinal fluid obtained which presented the same features, i.e., a uniform distribution of blood in all specimens, the failure of this blood to coagulate on standing, and the appearance of xanthochromic cerebrospinal fluid after centrifugalization, as that examined six months previously. Hematological studies were similar to those observed at the time of his first admission. The patient again made an uneventful recovery and sixteen days after admission was discharged from the hospital.

When seen six years later, and at the age of seventeen years, the patient was in excellent physical condition, had made satisfactory progress in school, and had not had a recurrence of his symptoms. Reëxamination of the blood did not reveal any abnormality other than a slight anemia and a slight reduction in the number of platelets; hemoglobin 93 per cent; red blood cells 4,390,000; white blood cells 10,200 with a normal differential count; bleeding time 3 minutes; coagulation time (venous blood) 8 minutes with a firm, retractile, normal clot; fragility test normal; reticulocytes normal, and platelets 170,000.

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ESSENTIAL PUBLIC HEALTH SERVICES*

BY KENDALL EMERSON, M.D.†

THE American Public Health Association devotes much of its attention to the study of standard practices in public health work and to the appraisal of the value and efficiency of their administration as currently carried out by state, municipal and rural health departments. Needless to say, widely divergent procedures are encountered in the various parts of this large country. Such a situation could scarcely be otherwise in a union of forty-eight separate and temperamental states to which the federal government has delegated practically complete autonomy in the exercise of most police powers, including protection of the people's health.

Municipal and local health authority is usually delegated by the state, the latter retaining more or less supervisory control. Here again the widest variation in actual practice occurs. For example, the neighboring and fairly comparable states of Pennsylvania and New York present instances of highly centralized control on the one hand and widely distributed authority on the other, with, in the second instance, much effective power still applied through the use of subsidies. The relative merits of divergent practices is a difficult matter to appraise since health conditions appear to remain tolerable despite the prevalence of one or another of several varying procedures. Furthermore, there is much difficulty in proving the greatest economy in the application of any single plan, especially as records are frequently so kept as to render comparison unsatisfactory.

With this preamble we must admit that standard practice for one municipality does not of necessity point the way to effective health service in another. Averages are of the greatest benefit in studying the problem in the large. But when averages are brought into an argument we are at once faced with the naïve in-

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formation that the particular city under discussion is an exceedingly exceptional municipality, that you cannot do there many things that might be quite appropriate for certain other inferior localities, but that would never fit the exquisite requirements of this individual city.

During the alarming raids on health budgets which took place last year the Public Health Association issued an official declaration of attitude on desirable standard minimum functions and suitable organization of health activities. The standards were admittedly minimal and are helpful rather as setting a level below which the danger point is reached than in pointing out a reasonable upper limit either in activities or expenditure. The outstanding principles can be summarized in a few words, — adequately trained leadership with full responsibility for accomplishment laid upon the administrative officer, expenditure of at least $1.00 per capita exclusive of hospital services, freedom from political interference for the health officer and liberty to select his own trained personnel. The two comprehensive objectives of a health department laid down are the control of communicable diseases and the promotion of child health. As indispensable instruments for the accomplishment of these objectives are vital statistics, laboratory service, environmental sanitation, public health nursing and a general program of public education in personal and community health.

All of the health activities of a city need not actually be performed by the health department, but it should be held responsible for recognizing and prescribing the services needed, for participating in and guiding the broad general health plan of the city, for insuring the coördination of all health activities carried on by other agencies, official and voluntary, for an adequate program of health service applicable to and divided fairly among the whole community. A frequent misconception prevails that the health department is for the *poor* of a city. Nothing could be more untrue. It serves every citizen just as impartially as does the police or fire department. Could the people at large be brought to realize this fact more clearly, their sympathy and support would be more readily forthcoming. To teach them is one of the functions of health education for the people at large, a function for which busy health departments are none too well equipped, but one where volunteer agencies have done yeoman service in the past, and which they are continuing at the present time. Meanwhile more rational systems of health education are creeping into the schools and colleges and it is hopefully believed that popular interest in an adequate community program of preventive medicine can be progressively developed through general educational channels.

With the mass of public health knowledge and experience now available it is a fairly simple matter to set up what appear to be adequate programs. Statistical studies can demonstrate what accomplishments are possible under forced draught as in the case of the Framingham experience with tuberculosis or the Cattaraugus County and Syracuse projects. In other words the things to be done and the results to be expected are not so difficult to define. The hitch comes in the administrative set-up appropriate and acceptable to any given city. As is the case in state health departments, one city shows excellent results with a strictly centralized plan, another districts its territory and establishes health centers to which local responsibility is delegated. Both procedures have their advocates and while I believe the district principle is far preferable, yet in reviewing the activities of a given health department working successfully from a single center one might hesitate to suggest a radical decentralization. Unquestionably economy of operation is a prime desideratum, especially in these critical days.

On one point there is very little room for dispute. We cannot have either an efficient or economical health department unless it is staffed by trained personnel. It is only necessary to allude to the grievous harm done to the public health service of America by the interjection of politics into our system of official appointments. To an Englishman it seems a strange situation that we should be willing to put in jeopardy so precious a thing as the public health at the arbitrary dictation of uninformed politicians. Unfortunately some of our leading cities are seriously affected with this virus. Whatever other benefits may result from the recent political convulsion in New York City the revolution already effected in the health department would be enough to justify its being considered a success.

What are the qualifications of a good municipal health officer? Well, education and training are indispensable. Whether he should have a medical degree is still debated among us, though this question has been settled in the affirmative in practically all other countries. That he should have no political entanglements is self-evident. Passing over many other desirable traits there are two which seem to me indispensable, he must have talent as an *organization worker* and he must be able *to utilize effectively not only the resources of his own department but all the health resources of the community,* including first of all the medical, nursing and dental professions, as well as volunteer health agencies and committees, the department of education, hospitals, industrial organizations, and social agencies. This is no task for an amateur or for one with any motive other than that of unselfish service to his community. It is introducing an overworked word

but the idea conveyed in "statesmanship" comes nearest to a comprehensive description of the basic equipment necessary to successful administration. That such standards are not set too high is proved by the fact that there are many men in charge of city health work today who fulfill them, although their number is vastly less than it should be. Good generalship is said to be just as important in conducting a retreat as in planning an advance, a significant fact in the face of present retrenchments.

With the understanding that effective health administration depends more on the administrator than on too stereotyped methods of administration we may proceed to discuss in a little more detail the essentials in public health procedure. The historical approach may lend clarity to such discussion.

At first thought vital statistics might not appear to have claim to historical priority. In early tribal life a patriarch's wealth was estimated by the number of his flocks and herds. Likewise the power of the tribe itself was recognized as co-equal with the number of its human components. For survival a high ratio of births must be maintained and the injunction to be fruitful and multiply was doubtless originated by some early statistician who, like his successors through the ages, revelled in an opportunity to play the rôle of alarmist. Long before systematic death records were kept, the numbers dying during exceptionally severe pestilences were recorded and have come down to us through history. Without question the preservation of the fighting strength of armies was among the reasons for the early development of more regular and accurate vital statistics. Imperfect as such records are, prior to comparatively modern times, they still furnish us with invaluable data in any review of man's rise through the various stages of civilization, which parallels his gradual appreciation of the value of a human life.

To-day even more accurate vital statistics, birth, death and morbidity rates are recognized as at the basis of all scientific advance in public health. Without such records research would be idle and fruitless. It would appear self-evident that historically and practically the keeping of such painstaking statistics, as well as equally accurate records of work done in all departments of the service, is a basic function of a health department and that lessening of this wisely supervised activity would lead to inevitable lowering of health standards and health protection for the community.

The fact that certain diseases are communicable was recognized and recorded by Hippocrates. Methods of *preventing* infection, however, remained largely in the realm of superstition until the chance discovery during the middle of the nineteenth century of the connection between dirt and disease, at which time the term "filth diseases" was coined. Historically, then, environmental sanitation appears to rank next to vital statistics in the development of a science of preventive medicine. From that significant discovery has developed the profession of sanitary engineering, one of the two outstanding factors in the control of preventable disease.

The second factor of like significance and appearing a few years later, was the establishment of the public health laboratory which followed upon the discovery of the bacterial origin of communicable disease. Through its epochal discoveries bacteriology has made possible the identification of the causes of most infectious diseases and has already devised specific measures for combating many of them. Such discoveries could never have rendered their outstanding service to the community save through the medium of the public health laboratory.

If there are four basic functions of the health department which could not safely bear curtailment in their high professional conduct, I believe you will agree they are administration, vital statistics, sanitation and laboratory service. It is unthinkable that any enlightened community should consider crippling such essential contributors not only to its happiness and welfare but to its very existence.

In our historical review, then, we may say that scientific research, plus administrative experience, has supplied us with the means for adequate protection of the people's health. Next comes the practical application of those means. That task has fallen upon the professional health administrator or health officer. His allies are two, the police powers delegated to him by legislation; and public education in the laws of personal and community hygiene. From the days of the medieval lazaretto and the more modern pesthouse the use of the police force in administering public health has gathered a disrepute which makes its usefulness decidedly problematical. Occasionally, rather than currently, it is invoked in our modern civilization. Persuasion and the more gradual method of education have superseded it to a large degree, sometimes with the effect of slowing up progress temporarily but on the whole, it is believed, for the ultimate public good. An instance of how it may delay accomplishment is that presented by tuberculosis, where the failure of compulsory isolation certainly causes a serious lag in preventing the spread of the disease. But this is probably offset by the vastly greater efficacy of our sanatoria under a voluntary plan than would be possible under one of unwilling restraint.

It would appear to be self-evident that the problem of applying an effective public health program without undue coercion must depend on developing a *receptive mental attitude among the people*. This task has been from the first one of the heavy burdens on health depart-

ments. Fortunately they have had assistance. Despite its preoccupation with curative medicine, the medical profession itself has been the strongest prop on which the public health service has leaned in prosecuting its mammoth task. Of late the phrase "every doctor's office a substation of the health department", has gained currency.

At the same time it was recognized a generation ago by such men as Osler, Welch, Biggs and Trudeau that more help than the doctors could render must be found. Dr. Livingston Farrand speaking at the quarter-centennial meeting of the National Tuberculosis Association remarked that he saw as the greatest service rendered by the volunteer health agency the association in an organized health education movement of both lay and professional members. Through the development of such societies and their spread to all parts of the country it has been possible to reach the public far more quickly than could have been done through unaided official channels. Health officers through fear or obstinacy have failed to take full advantage of this fruitful source of assistance.

More recently departments of education have awakened to their obligation and opportunity in matters of health education. Curricula are being devised and somewhat slowly introduced which it is believed will definitely raise standards of health knowledge among the younger generation now in the school rooms of the country.

Meanwhile the great body of nurses, both private duty and more especially those in public health, are the practical teachers as they bring their training and experience to bear on the families with whom they hold such intimate contact. In a sense they are the clinical teachers and for that reason their service is of inestimable value.

I need only refer to the health instruction developed by *the large industrial interests, the life insurance companies, the health committees of various clubs and societies, both national and local.* This is a heartening array of allies for the public health service if it will but make full use of such resources under its own safe guidance and control. Phillips Brooks once said that he never did anything himself that he could get someone else to do for him. That left him only the things which he alone could do. This principle may perhaps be utilized more than at present by a health department crippled through budget restrictions.

It is more than likely that other important functions of the health department may be found, the burden of which, like health education, might be shared by agencies outside the official budget. Take for example the vexing question of clinics. Free treatment clinics for the indigent appear to be accepted as part of our social welfare program. They are provided by hospitals and in some instances by public health departments. I see no indication that they should cease to be a proper burden on the taxpayer. Diagnostic and child hygiene clinics are in another category. They are justly considered a potent means for the early discovery of infectious disease, and, therefore, especially effective as centers for popular health education, and a most valuable factor in preventive medicine. To abolish them would be a retrograde step of the utmost gravity. On the other hand the expense of maintenance is considerable. Cannot some method be devised for relieving the public health service from carrying the whole burden? This I leave merely as a question. I know nothing of the Boston situation in this respect, but submit that it is one which might well receive the careful consideration of District Medical Societies in their social planning, including emergency and long time planning. Temporarily the medical profession may make further contributions awaiting permanent solution, thereby keeping the clinics running.

Then there is the matter of research. We have seen that upon scientific research is based all of our advances in modern public health practice. Much of this has been done by the public health service itself, in laboratories, through surveys, appraisals, demonstrations and the like. Its value has been and is inestimable. That it should cease or be curtailed is not to be considered. The question arises again, however, as to whether there are other community agencies which might take over some of the load. This suggestion sounds strange coming from one who knows what priceless contributions the pathological laboratory of the City of New York has made to public health knowledge under the brilliant direction of Dr. William H. Park. Yet even so outstanding an example still leaves it possible to ask if university laboratories, expensively equipped as they are and often well endowed, could not take on more of this research work. It is dangerous to bring up the situation in the health department of Indiana, since its dismemberment under a too radical governor has met with little but criticism. However, transferring the public health laboratory to that of the State University, also supported by tax money, has apparently worked tolerably well and has greatly lessened the cost to the State of maintaining two completely equipped agencies of the same nature.

I understand that Boston is pulling up her belt like the rest of the world, that public appropriations are showing a somewhat sickly mien here as elsewhere and that economies are prescribed for the health department. As usual she took the sane and intelligent step indicated by the circumstances and appointed a committee of review to advise on the possible lopping

off of obsolete functions and safe restrictions in expenditure. Sanitary service is the oldest and therefore may be a good place to look for barnacles. A world-wide search could not have produced a happier choice of personnel for the members of that committee. Quite as a private comment let me remark that it was a source of astonishment to some of us down on the little island where I now reside that your Mayor should possess the temerity to challenge certain of the committee's recommendations.

I am of course incompetent to discuss Boston's housekeeping arrangements and should not be so indiscreet as to make the attempt. I may, however, quite fittingly report to you the opinion in which your broad health plan for the city is held in other places. To us it has always appeared that the gift of your distinguished citizen, Mr. George Robert White, is one of the most outstanding civic services of which we are aware. The eight health centers which you have established are looked upon as the basis on which it is possible to build the ideal preventive medical program for a large city. We have watched their development with sympathy and interest and have envied you their possession.

As we understand them, they are the actual clearing houses through which united action of official and voluntary health and social agencies are enabled to bring about equal distribution of welfare work to the community in the most efficient and economical manner. Through them it is possible to furnish certain types of medical service such as diagnostic clinics and those for the treatment of the indigent, a very large amount of public health nursing service and necessary sanitary inspection. And perhaps most important of all they are the ideal centers for popular health education.

When we add to this unique system the exceptional health services rendered the city by the Boston Dispensary, the Lying-In Hospital, the City Hospital and the sanatoria for chronic disease, the Boston Health Department is seen to be blessed far above those of other cities in the equipment provided through which it is enabled to lay out and execute an adequate health program for all the people.

Yet equipment alone is not sufficient. Bricks and mortar will not by themselves either cure or prevent disease. They are but the raw materials of the health structure. To give them life and power to fulfill the vision of their creators two factors are indispensable, adequate financial resources and well-trained personnel. Of the latter there is no lack in Massachusetts. Your task is merely one of selection. For the former, taxation is no doubt the principal source and at the moment the source runs low.

How to do things that cost money when money is lacking is a problem that I have studied prayerfully for many years, both in my private life and in my public relationships. The riddle is not solved as yet. However, sometimes a friend will give you a dinner even when he cannot spare cash. This is a moment when public health needs friends. Here in Boston you are vastly fortunate in that you have an enlightened medical profession cordially inclined toward your public health service. Have you exhausted the possibilities of advice and possible assistance from organized medicine?

There is no city where the health and welfare activities are more carefully, and doubtless economically, coördinated than here. Were I elsewhere I could give no better advice than to follow Boston's lead in this direction. I fancy that volunteer aid from health agencies is already at the disposal of the official department to its fullest extent.

The universities and the hospitals would appear to offer some fields at least for exploration. Is it possible that the Minneapolis resident interne plan of which we are now hearing good reports would have any application here in caring more economically for the indigent? As you see I can but ask questions. Their answers must of course be given in the light of your local situation. These and many others, that might be proposed, may open up avenues of discussion whether profitable or otherwise.

Meanwhile it is of the utmost importance that the people of the city be kept informed of the dilemma in which the public health department is placed by the apparently necessary reduction of appropriations for health and that in particular the members of the city council be acquainted with the grave responsibility which they assume when health budgets are reduced below the accepted minimum.

In conclusion, we have seen that there are certain basic functions of a public health service that can only be carried on safely and effectively by the service itself. We have also examined some of the broader functions of a health department, not questioning their desirability but from the point of view of the gruelling economy necessitated by to-day's conditions. When drastic reductions are ordered and the budget cut twenty to thirty per cent, the health officer is faced with two alternatives after he has cut salaries to the bone, eliminated obsolete activities and supernumerary personnel. On the one hand he may maintain all his departments and services, making a horizontal cut in each and thereby limiting the number of personnel and the extent of service possible throughout all branches of the work, or he may curtail or eliminate certain less vital departments allowing the essentials to continue unscathed. The latter choice seems to me the saner. Then the health officer who is at the same time

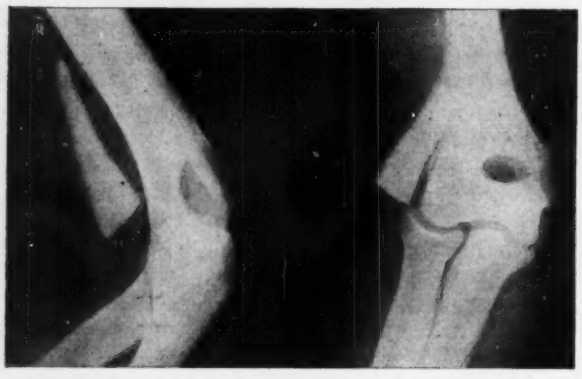
a statesman will cast about to see where in other public services such as the schools, hospitals or welfare groups he can discover agencies to whom some at least of the work can be transferred. Nor will he stop there, for there still remain certain volunteer organizations on whom he may call for temporary aid. And lastly there is Uncle Sam himself who has come to the rescue during the past year with his civil works authority and who, if one reads the signs of the times aright, may perhaps be cajoled into continuing in loco parentis for a considerable period yet to come. In the last analysis the heaviest burden must be borne by the administration. There are times when almost superhuman skill and judgment are needed to weed out nonessentials, to preserve basic functions and to foster those in particular which point with most promise to effective accomplishment in the long look ahead.

FOREIGN BODY IN ARM

BY JAMES WARREN SEVER, M.D.*

IN these days of automobile accidents, associated with cuts from broken glass, it is not unusual to find small pieces in a wound. The following case is of interest for two reasons: first, because such a possibility was wholly overlooked; and, secondly, on account of the large size of the piece of glass.

This man, aged 36 years, a carpenter by trade, was in an automobile accident in Nova Scotia on September 14, 1926. The car tipped over, and the windshield was broken. He sustained a small transverse cut on the outer side of the left arm



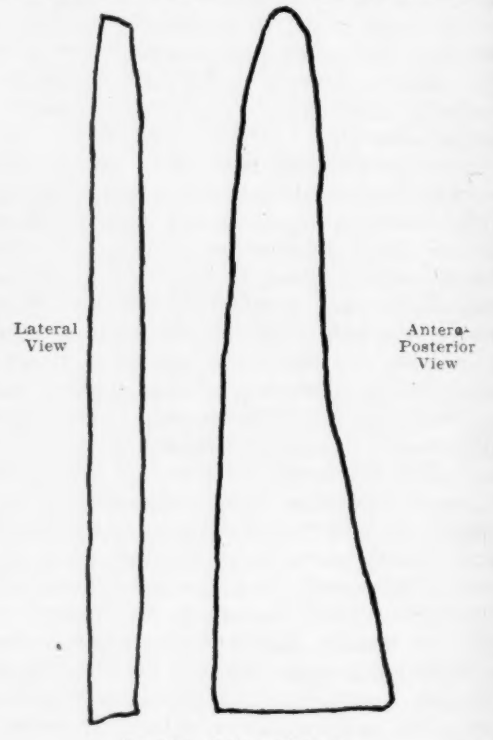
just below the elbow joint on the outer aspect. The wound was cleaned and closed by two skin stitches, by the local physician, and healed promptly by first intention.

After coming back to Boston, his arm bothered him some, and he saw his family physician, who advised an x-ray which was taken October 4, 1926. (See x-ray.)

This x-ray was interpreted as showing a large loose piece of bone, apparently, broken from the lower end of the humerus, and lying to the outer side and above the head of the radius.

Clinically, this piece could be felt, as a movable object, causing slight tenderness and interfering with the motion of the elbow. The arm could be flexed only to 75 degrees and extended only 125 degrees at the elbow. There was no redness or swelling. The original wound was healed. Operation was advised for removal of this piece of supposed bone, as it interfered with function, and his ability to work. This operation was done October 16, 1926.

Operation: Incision about three inches long was made on the outer side of the lower end of the humerus, and carried down to what appeared to be in the x-ray a fragment of bone. This fragment turned out to be a large, thick piece of glass, apparently from a windshield of an automobile. It was 3½" long, 1" wide, at its lower end, and ½" wide, at its upper end. It was ¼" thick throughout. (See tracing of fragment.) It was lying beneath the



Tracing of piece of glass.

lower end of the biceps muscle, the upper end extending under the brachial artery and in contact with it. The lower end was underneath the supinator longus muscle. It was easily removed in one piece, as well as several small pieces of glass, which could be felt elsewhere in the wound. The wound was closed and it healed at first intention.

When last seen, this patient had normal motion in the elbow joint, and has returned to work.

The points of interest in this case are, first: the total lack of knowledge on the part of the patient, and the original physician, that there was any condition existing other than a small cut on the arm, which healed promptly, in spite

*Sever, James Warren — Assistant Professor of Orthopedic Surgery, Harvard University Medical School. For record and address of author see "This Week's Issue," page 467.

of there being a large piece of dirty glass in the arm, which had passed through this opening. The original wound seemed far too small, when I saw it, to have allowed the entrance of such an object. Secondly: The confusion in regard to the x-ray interpretation. There was apparently such a large percentage of lead in the glass, that it threw a shadow in the x-ray films, practically equal to that of the bone, and so resembled a piece of bone. It was so interpreted, although there did not seem to have been any loss of contour to the shaft of the humerus, from which it could have been detached. Thirdly: The lack of sepsis and irritation, which this large fragment of glass, with a sharp point and edges caused, buried in the tissues, and subject to possible irritation from motion of the elbow joint. The fact that its sharp, pointed upper end was in contact with the brachial artery, led to the thought of possible puncture of that vessel at no distant time. Fourthly: Perfect restoration of function following removal.

A CASE OF REGIONAL ILEITIS

BY KARL T. PHILLIPS, M.D.*

A TWENTY-FIVE year old, married, cotton mill worker entered the Day Kimball Hospital on October 6, 1933, complaining of recurring stabbing pains in the lower right abdominal quadrant. The pain was just inside the scar of a previous appendectomy. He also had nausea, vomiting, dysuria, nocturia and general weakness.

The beginning of abdominal pain was in 1928. At this time he had gradual onset of slight dull pain in the lower right abdomen, just above Poupart's ligament. It never disabled him and would last only five minutes, occurring three times a day. The frequency of the attacks was variable but seemed to have a relation to the taking of meat. Their severity or frequency did not increase. Stools were regular and normal.

On the night of August 31, 1932, fourteen months before the present admission he was awakened by a severe abdominal cramp and was sent into the hospital the next day. On admission his temperature was 99.9, pulse 100, respirations 24. An appendectomy was done three hours after his admission. The appendix was reported as reddened and long and there was considerable serum in the peritoneal cavity. No other findings were noted. He had no loose stools while in the hospital and was discharged on his fourteenth postoperative day. He felt perfectly well after this until the beginning of his present illness.

Five months before admission he first noticed a dull ache in the lower right abdominal quadrant. This continued for two months. Then sharp stabbing pains had occurred, becoming more severe, particularly after meals. He had had dysuria for four months, not increasing in severity. His stools, which had always been regular, had for the past four months moved only with physic. He had lost twenty pounds in two and one half months. For two weeks he had had an ache in the right groin which radiated up into the right renal region. He vomited four times during the week previous to admission and he had had slight nausea, bloating, belching and heartburn.

Physical examination on admission on October 6, 1933, revealed him to be fairly well developed but poorly nourished. The abdomen was slightly distended and tympanitic throughout. There was a well healed low right rectus scar. Just to the left of the umbilicus there was slight tenderness but no spasm. There was also slight bilateral renal tenderness. Rectal examination and the rest of the general examination were negative. The temperature was 97.8, pulse 76, respiration 18, w.b.c. 11,500, r.b.c. 5,100,000, hemoglobin 80 per cent. The differential count was 76 per cent polymorphonuclear leucocytes, 2 per cent large lymphocytes, 22 per cent small lymphocytes. The urine was negative.

The working diagnosis was partial intestinal obstruction from postoperative adhesions, with reservations in regard to the right kidney.

X-rays of his gastro-intestinal tract were reported as showing vigorous peristalsis, considerable pyloric spasm and delayed emptying of the stomach. No ulcer was seen. The six-hour film showed all the barium in the large bowel.

Retrograde pyelogram on the right was negative as was the urine from the right kidney.

During the eleven days following his admission his temperature and pulse were normal. For the first time he had two loose stools daily. The abdomen was slightly distended and two or three times a day he had severe cramp-like pains in the right lower quadrant, at times requiring morphia to relieve them. Vomiting occurred twice during the first forty-eight hours but not afterwards.

On October 16 there were tenderness and spasm in the lower right abdomen and with it a sudden increase in the severity of the pain. He was operated upon on October 17. A low mid-line incision was made. A large amount of serous peritoneal fluid was found and a five inch segment of terminal ileum that was twice its normal size, bluish red, and covered with a thin fibrinous exudate. This felt solid and was nodular. It was freely movable with no adhesions to surrounding viscera. The mesentery of the involved portion was packed with firm enlarged glands. The ileum above was three times its normal size and there were two inches of pale but normal gut below extending to the cecum.

The diseased ileum was resected and after the proximal lumen had been sutured down to a size to match the lower, an end-to-end anastomosis was performed. An enterostomy was done in the distended gut above and a rubber tissue drain inserted alongside of the enterostomy tube.

Examination of the segment removed showed a lumen the size of a lead pencil. Pathological report was as follows:

"The serosa is smooth and glistening. Scattered over the serosal surface are accumulations of dark purple blood. The wall of the ileum is thickened and pearly white. The mucosa is dull and roughened and thrown into many folds in which are seen purple hemorrhagic areas.

"Microscopic examination: — The surface epithelium is poorly preserved in places. In an area where it is missing the surface is covered by fibrinopurulent exudate which extends into the tunica propria. Some cellular infiltration of polymorphonuclear leucocytes, small round cells and large mononuclear leucocytes is prominent in the tunica

*Phillips, Karl T.—Adjunct Surgeon, Day Kimball Hospital, Putnam, Conn. For record and address of author see "This Week's Issue," page 467.

propria. There are accumulations of small round cells to lymph follicle formation with marked overgrowth of germinal centers present in the submucosa. There is some edema of the muscular coat. In a lymph node in the mesentery there is some overgrowth of the germinal centers. Another preparation shows marked ulceration of the mucosa and edema of the tunica propria, submucosa and muscularis. Aggregations of small round cells are frequently seen in the submucosa. Another preparation shows similar structure. No areas of caseation necrosis or giant cell formation are seen. The accumulations of small round cells in some areas suggest early tubercle formation. This, however, cannot be definitely stated as no areas of caseation necrosis or giant cell formation are noted anywhere in the preparation.

"Diagnosis: Ileitis."

His convalescence was stormy. The enterostomy tube was pulled out by the patient on the second postoperative day. On the twelfth day his wound had to be opened digitally under gas-oxygen anesthesia to drain a walled-off abscess in the ileocecal region. On the seventeenth postoperative day he developed a urinary fistula and a catheter was inserted through his urethra and retained. Stools were liquid and frequent from the fourteenth to nineteenth days. After this they were normal. The fecal fistula closed ten days after this and the urinary fistula closed on his thirty-ninth postoperative day.

On the one hundred and fourth day after admission, his wound was healed, his general condition was much improved and he was gaining weight rapidly. His stools were normal and he was free from pain.

The serum present at the time of his appendectomy makes one suspect that symptoms from ileitis started at least thirteen months before his admission. The pathological report of the appendix in 1932 was chronic appendicitis.

Since his discharge from the hospital his fecal fistula has broken down with recurrence of visible peristalsis and abdominal pain. On May 3, 1934 a side-to-side anastomosis was done between the terminal ileum and the transverse colon, and he is making a good recovery.

This case is reported because of the recent attention paid to this illness and the lack of prominence of the loose stools which usually accompany it.

THE MEDICAL SECRETARY CURRICULUM AT WESTBROOK JUNIOR COLLEGE, PORTLAND, MAINE

BY RUTH E. IRWIN, M.A.*

IT is generally agreed that there are many occupations which require more training than can be provided for in our high schools, but which do not require the four years of training of the college type. These occupations are often referred to as "semi-professional". One investigator lists fifty-six semi-professions and occupations for which two years of college work will furnish adequate preparation. With the growing uncertainty and need for economic security in professional occupations many students are turning toward courses in the curriculum offering specialized training and are choosing courses which, through practical application in the classroom, give tangible evidence of progress and skill.

In a questionnaire survey of the interests of over 400 girls graduating in June, 1933 from the high schools of Portland and vicinity, it became evident that one girl out of every four was interested in the semi-professions allied to medicine. After careful consideration and with the advice of a number of physicians, it was decided to include in the Westbrook Junior College program of studies a two-year terminal curriculum for the training of young women to work in doctors' offices. The purpose of the course is to train students of high social, manual, and verbal intelligence to handle the intricate details in the busy doctor's office leaving the doctor free from the cares of the office routine. Their training is threefold: secretarial, technical, and cultural. They will be expected to take dictation, fill out case records, file medical reports, investigate the social and financial status of the patient, prepare for and aid in minor treatments, and attend to the routine analyses.

In the spring of 1933 a group of Portland physicians was consulted and Dr. Adam P. Leighton, then president of the Cumberland County Medical Association, was asked to choose an advisory committee from the association to act as consultants with the junior college committee in planning the program for the Medical Secretary Curriculum. Dr. Milton D. Proctor, president of the Westbrook Junior College, and Miss Ruth E. Irwin, director of the Medical Secretary Course, met in consultation with the committee appointed by Dr. Leighton, which consists of the following members:

Dr. Mortimer Warren, pathologist, Maine General Hospital.

Dr. Edwin W. Gehring, then president-elect of the Maine Medical Association and chief of the Medical Service, Maine General Hospital.

Dr. Frank Smith, practising physician, Westbrook, Maine.

Mrs. Maud Staples, Director of Nurses, Maine General Hospital.

All students working for the medical secretary certificate are to come before another committee approved by the C. C. M. A. at the end of the two-year period for a final examination.

The following topics cover the major part of the technical subject matter included in the medical secretary curriculum:

*Irwin, Ruth E.—Westbrook Junior College 1932-. For record and address of author see "This Week's Issue," page 467.

CHEMISTRY:

Introductory inorganic and organic chemistry.
Study of important elements.
Solutions.
Use of metric system.
Foods.

Physiological chemistry.
Gastric analysis.
Urinalysis.
Specific gravity, sugar, albumen, blood, microscopic.
Blood.
Total red and white cells, hemoglobin (Tallqvist and Sahli), differential.

HEALTH:

Anatomy, physiology, hygiene.
Pathology.
Dietetics.
Scope of public health work; National, community, hospital, research laboratories.

BACTERIOLOGY:

Methods of sterilization.
Preparation of culture media.
Technique of plating.
Methods of studying bacteria.
Technique of staining.
Preparation of staining solutions:
Loeffler's Blue, Sterling Gentian Violet, Lugol's iodine solution, Neisser's Stain, Ziehl-Neelsen, Carbol Fuchsin.
Study of pathogenic microörganisms, immunity, antitoxins, vaccines.
Identification of more common pathogenic forms:
Staphylococci, Streptococci, Pneumococci, Tubercle bacilli, Pertussis bacilli, Gonococci, Meningococci, Influenza, Typhoid, Paratyphoid and Colon.

OFFICE TECHNIQUE (MEDICAL):

Aseptic technique.
Preparation and sterilization of supplies; swabs, gauze pads, gauze sponges, gloves.
Identification of surgical instruments.
Setting up and aiding in minor treatments.
Bandaging.
Draping of patients.
General massage.
Blood pressure.
Temperature, pulse, respiration.
Materia medica (condensed).
Weights and measures.
Pouring solutions.
Preparation of antiseptics.
Use and dosages of more common drugs.
Symptoms of overdosage and antidotes.
First Aid measures.
Cleaning up after treatments.
Observation of hospital technique.
Preserving specimens.

OFFICE TECHNIQUE (SECRETARIAL):

Medical correspondence, bills, reports.
Case reports.
Medical indexing and filing.
Preparation of medical manuscripts.
Medical terminology.

PROFESSIONAL ETHICS:

Social case histories, financial status of patient.
Professional dignity.
Psychology in relation to sickness.

The curriculum is organized as follows:

First Semester		FIRST YEAR	Second Semester	
Hours per week	Credit hours		Hours per week	Credit hours
3	3	English	3	3
3	3	Community Life	3	3
6	3	Chemistry	6	3
4	4	Shorthand	4	4
4	2	Typewriting	4	2
2	1	Orientation	2	1
2	1	Physical Education	2	1
	—			—
	17			17
		SECOND YEAR		
3	3	Business English	—	—
—	—	Human Conduct	3	3
3	3	Hygiene	3	3
5	5	Secretarial Practice	—	—
8	4	Bacteriology	—	—
—	—	Medical Laboratory Technique	15	9
2	1	Orientation	2	1
2	1	Physical Education	2	1
	—			—
	17			17

At the present time arrangements are being made with the Maine General Hospital for the use of its facilities in pathological work and for the attendance of medical secretary students at some of the medical lectures in the nursing school. Arrangements are also being made with other medical organizations in Portland to provide practical experience for the students in various phases of the medical environment.

This is the first year of the two-year program. There are at present eighteen students enrolled in the course*. The constructive criticism and approval of members of the medical profession will be welcomed. There are two outstanding facts to be faced squarely: the overcrowded condition in the medical field and the disapproval by some doctors of the performance of technical procedures by assistants. The general opinion seems to be, however, that there is a real need in the field of medicine for a trained worker who can combine the qualifications of the nurse, technician, and secretary. Sincere interest and encouragement have been manifested by leading doctors and nurses in New England and in New York City. The Medical Secretary Curriculum, so far as its organizers know, has never before been part of any junior college program. The attempt has been made to select girls of pleasing personality, good academic rank, and potential skill, who may acquire not only technical training but also the benefits of association with girls of other interests in social activities on the college campus. The enthusiastic application and social adaptability of the group this year have been truly gratifying.

*September 1, 1934.—We already have eighteen more for 1934-1935 making a total of thirty-six first and second year students.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 20361

PRESENTATION OF CASE

First admission. A forty-seven year old married Italian cobbler entered for the first time complaining of weakness, dyspnea and epistaxis of four months' duration.

Approximately five months before admission he began to tire very easily while at work and often was forced to stop and go home and rest for a few hours. On some occasions he would remain at home for the entire afternoon. At the same time he also began to have frequent headaches in both the occipital and frontal regions, and often felt slightly dizzy, although he did not fall or stagger. Three months before admission he became definitely weaker and developed shortness of breath with the slightest exertion. With this his heart would race and pound. He developed edema of his feet and ankles, more marked at night. A physician told him that his heart was normal and that his blood pressure was 130. He was put on an egg and milk diet for fifteen days, after which he felt better and returned to work. He then began to have nosebleeds which lasted almost half a day. In the past three months he had had four of these episodes. Bleeding from the gums was frequent but never very marked. One month before admission the edema and weakness became more marked and his skin paler and even yellowish. He developed some puffiness around his eyes, usually more marked in the mornings. His doctor placed him on "liver treatment" and also gave him frequent injections, as well as medicine by mouth. It was later determined that he had received thirty to thirty-five intramuscular injections of liver extract (Parke Davis) in 2 cubic centimeter doses. Five days before admission he had his teeth cleaned. This was followed by a moderately severe bleeding episode. Three days before admission a severe and copious epistaxis occurred which continued without stopping until admission. He became very weak, could not take food and began to cough. He said that he had looked pale as long as he could remember. He had had no hemoptysis or hematemesis. His bowel movements had been black for the past month, but not definitely tarry. He had passed no frank blood by rectum. There had been no hematuria or smoky urine. He did not believe that he had lost more than three or four pounds in weight during the past three months.

His family and marital histories are irrelevant.

Occupational history. He had always been a cobbler, and for the past seven years had cemented leather soles with a rubber cement that might have contained benzol.

Physical examination showed a large, fairly well nourished middle-aged man lying in bed in no acute distress. The skin and mucous membranes were very pale. A few small purpuric spots were observed in the right axilla. There was mild pyorrhea. A small trickle of fresh blood was seen in the nasopharynx and on the gum margins. There were blood crusts in the left nostril. The right pupil was slightly larger than the left. Both reacted poorly to light. There was a right lower facial weakness. The heart was moderately enlarged, the left border of dullness being 10 centimeters from the midsternal line. The blood pressure was 140/70. The pulse was 120. There was a blowing systolic murmur all over the precordium and a faint early diastolic blow over the base and at the apex. The lungs were clear. The liver and spleen were not felt. There was slight edema of the ankles. There were no palpable glands.

The temperature was 100°. The respirations were 30.

Examination of the urine was negative. On admission the blood showed a red cell count of 1,050,000 with a hemoglobin of 30 per cent Tallqvist. The cells showed moderate variation in shape, slight anisocytosis and some polychromatophilia. The white cell count was 1,400, with a differential of 29 per cent polymorphonuclear neutrophils, 47 per cent lymphocytes, 2 per cent large mononuclears, 3 per cent metamyelocytes, 8 per cent myeloblasts and 2 per cent unclassified. The platelets were very few in number. There were 4 per cent reticulocytes. The volume index was .76. A Hinton test was negative. Examination of the stools was negative.

A sternal biopsy was performed, but the specimen was so minute that the amount of marrow available for study was inadequate. However, it appeared from the rather high proportion of stem cells and evidence of both red and white cell formation that the marrow was hyperplastic rather than aplastic. Both his red and white cell counts and platelets remained low. A smear on the eighth day showed 28 per cent myeloblasts and 13 per cent myelocytes. He had been receiving 5 cubic centimeters of nucleotide intramuscularly daily. His condition remained about the same, the red and white cell counts being practically unchanged. There was still a

little oozing from the gums. He was seen by several consultants, who agreed that nucleotide therapy offered the only hope theoretically and that transfusion should be used only as an emergency measure. He was given ten days of intravenous nucleotide therapy, 10 cubic centimeters three times a day. At the end of this period his red cell count was 1,150,000, the hemoglobin 35 per cent, the white cell count 3,100, with a differential of 31 per cent polymorphonuclear neutrophils, 30 per cent lymphocytes, 4 per cent large mononuclears, 14 per cent myelocytes and 15 per cent myeloblasts. The red cells showed marked anisocytosis and poikilocytosis and basophilia. Twenty-three per cent normoblasts were seen. There were no platelets. Nucleotide therapy was stopped on the thirty-fourth day. Ten days later a few petechiae appeared on the abdomen, the tongue and in the left conjunctiva. He was discharged on the forty-seventh day and told to return in two weeks for further treatment. He was put on ferric ammonium citrate 30 grains three times a day.

History of interval. He remained at home for a week, during which time his condition remained essentially unchanged. Two days before admission the bleeding from his gums and tongue returned and persisted. His appetite remained fairly good, but his strength gradually failed, forcing him to remain in bed most of the time. He took his iron as prescribed. He had no fever, cough or chills, but had difficulty in keeping warm. He had no vomiting. There was no loss of blood by rectum. A small area of induration which was present on the right buttock when he was discharged became rather painful, so that he could not lie on his back without discomfort.

Second admission, six days after his previous discharge.

Physical examination was essentially the same as at his first admission. In addition however there were many moist râles throughout the right chest. There was a firm tender indurated mass 9 centimeters in diameter on the left buttock, showing no redness or fluctuation.

Examination of the blood showed a red cell count of 920,000 and a hemoglobin of 25 per cent. The smear showed marked variation in size and shape of the red cells and moderate basophilia. There were two normoblasts, one megaloblast and no platelets. The white cell count was 2,200, with a differential of 38 per cent polymorphonuclears, 48 per cent lymphocytes, 1 lymphoblast, 3 large mononuclears, 7 myelocytes, 3 myeloblasts. Examination of the urine was negative except for a few red blood cells. The stools were tarry and showed a two plus guaiac.

Two days after admission he suddenly sat up in bed and developed air hunger and abdominal discomfort. His blood pressure fell to 90/60. His pulse rose to 150, his respirations to 48. He showed marked apprehension and some sweating. Rectal examination showed diffuse tenderness. The feces collected on the finger showed a strongly positive guaiac. He was given a transfusion of 600 cubic centimeters of citrated blood. Many moist râles developed in both chests. He died at 10 o'clock that night.

Differential Diagnosis

Dr. Wyman Richardson: I cannot explain the frequent headaches in the occipital and frontal regions, but I simply bring up the symptom because in a case I saw some time ago I did not pay enough attention to headaches in a patient with leukemia. It was later demonstrated that this patient had a severe leukemic meningitis. I should like to pay more attention to these headaches, but I assume they were not prominent because they are not mentioned later in the history.

There is no history of bleeding before the weakness appears.

I might say that the symptoms seem to me to be entirely explained on the basis of anemia.

The physical examination again shows essentially nothing but the presence of anemia. We are told that the pupils reacted poorly to light. In my experience such pupils are nearly always normal. The diastolic murmur can well be explained on the basis of a so-called hemic murmur. I do not think we have to assume that there is a definite valvular lesion. There was some question about that murmur anyway I gather.

I looked at two of the blood smears, which were undated, and in some respects I disagree with what was reported. One of these blood smears showed practically no normal lymphocytes, I think, and those that resembled lymphocytes seemed to me definitely atypical cells, not infrequently containing nucleoli and with cytoplasmic staining very different from what one would expect in a normal lymphocyte. Besides that there were many very young cells presumably of the white cell series which I did not attempt to classify further than that. The other smear showed more lymphocytes, apparently normal, but there still were present many undifferentiated young cells. The red cell description I agree with. There was marked variation in size, some in shape, some hypochromia, but still evidence of considerable red cell regeneration.

Whenever I see a note "no platelets" it makes me skeptical, because I think you ought to find at least one platelet in the blood smear in order to be sure the platelets are stained. However, in the smears that I saw the platelets were stained but were very, very scarce.

In the history of his second admission there

is a note that there was a large induration of the right buttock which was not fluctuant and not red, but was tender.

DR. AUGUSTUS S. ROSE: I might say that that was at the site at which the injections of nucleotide were given.

DR. RICHARDSON: I assumed that was the case. In regard to why they gave him nucleotide, I assume that they thought this might possibly be a granulopenia of idiopathic type.

Another point was brought out. This man was a cobbler and had been using rubber cement and possibly was exposed to the fumes of benzol, and, with benzol granulopenia, nucleotide might be of some benefit. The effect was not very marked, although the white cell count did come up.

Essentially the picture is the same at the second admission except that he is sicker, and then he dies rather suddenly. I do not think that a man with a red cell count of 900,000 and a hemoglobin of twenty-five per cent or less takes much of a hemorrhage to bring him beyond the critical level, and he may die from massive gastro-intestinal hemorrhage.

The differential diagnosis then includes perhaps quite a number of things. We might consider hemorrhage. It seems to me there is no history of hemorrhage. The picture is certainly not that of an acute hemorrhage. If it were chronic hemorrhage with a very tired out bone marrow one would not expect to have the evidence of red cell regeneration that is present or the presence of abdominal white cell forms to this extent.

So-called idiopathic aplastic anemia might be considered, but this picture is not that of a complete aplasia of the bone marrow. There is red cell regeneration, and furthermore there is the evidence of sternal bone marrow, which did show a small amount of bone marrow not suggesting complete aplasia.

This patient did not run the stormy course that patients with acute idiopathic agranulocytosis usually do. He showed no ulcerations and the effect on the entire bone marrow was at least as marked as the effect on the granulocytes themselves, if not more so.

In patients who die of benzol poisoning it is my experience that there is a steady drop in the white cell count down to a very low level. One would not expect to have this degree of aplasia without more effect on the white cells.

Purpura hemorrhagica is difficult to rule out. I ruled it out on the basis of the presence of numerous young white cells. Subacute bacterial endocarditis seems very unlikely. Pernicious anemia is ruled out by the failure of liver treatment. Also, the picture is not typical of it in any respect. Myelophthisic anemia from some other tumor is possible, but there is no evidence of any such thing.

So I come down to an acute leukemia as the most likely diagnosis. It fits in with the entire picture. The only thing against it is the absence of lymph glands, which I do not think is sufficient to throw it out. The appearance of very early atypical cells also fits in with this diagnosis. I make a diagnosis of acute undifferentiated aleukemic leukemia. If people think I am hedging in calling it undifferentiated I will call it lymphatic. I think he had a hematoma of the right buttock. I do not think that it was sarcoma or abscess, and I think the thing that pushed him over was massive hemorrhage from the gastro-intestinal tract.

CLINICAL DISCUSSION

DR. CHESTER M. JONES: This patient was on the ward about a month while I was on service. We followed the same line of thinking that Dr. Richardson has followed. I felt personally from the start that this patient had aleukemic leukemia, and I felt exactly as Dr. Richardson does in classifying it. I think we simply said aleukemic leukemia.

The question of treatment in this case was of some interest. We all felt while the patient was on the service the first time that treatment was going to be of no avail, but we had to make an attempt because we could not make a flat-footed diagnosis. There had been a history of benzol and that was one reason nucleotide was given. Another reason was that Dr. Jackson saw the patient after he had been on the ward two or three weeks. We decided to treat him with massive doses to see whether we could get any results rather than with any sure hope of giving relief. We did not transfuse him because we thought it would be a waste of money and work. Our experience in leukemia is that in any blood disturbance, except those following hemorrhage, transfusion retards bone marrow regeneration rather than stimulates it. I suppose the only reason it was finally done was to satisfy the family. The patient was discharged at the end with a diagnosis of aleukemic leukemia I think.

CLINICAL DIAGNOSIS

Aleukemic leukemia.

ANATOMIC DIAGNOSES

Aleukemic myelosis.
Bronchopneumonia, marked, right.
Pulmonary edema.
Pulmonary infarction.
Acute fibrinous pleuritis with effusion, right.
Chronic fibrous pleuritis.
Acute fibrinous pericarditis.
Petechial hemorrhages of the pericardium.
Thrombosis of the right internal iliac vein.
Operative scar, sternal biopsy.

Pathologic Discussion

Dr. Tracy B. Mallory: The autopsy showed comparatively little in gross except for a few things which tend to explain the final episode. He had, as you remember, acute abdominal pain. We found nothing in the abdominal cavity that seemed to account for that, but he did have consolidation of one of his lower lobes and an acute diaphragmatic pleuritis, also acute pericarditis, and there is little doubt that the abdominal pain was referred from the thoracic cavity. We also found a thrombus in the right internal iliac vein which extended part way up the vena cava, but so far as I could make out that played no essential part in his symptomatology.

The mass over the buttock unfortunately appears not to have been investigated.

The other findings at the autopsy were all essentially negative. The liver was normal in size. The spleen was atrophic, weighing only fifty grams.

The bone marrow throughout all the bones examined, which included vertebrae, ribs, sternum and the shaft of the femur, was red and hyperplastic. We were quite unable to make a diagnosis in gross and there is still considerable doubt as to the diagnosis, or at least as to the proper name to give it.

A moment's digression on the subject of bone marrow biopsies is perhaps worth while. There is no question that a proper bone marrow biopsy can be very valuable in many obscure blood diseases, for the circulating blood does not necessarily accurately mirror the conditions in the marrow where the true pathologic lesion lies. It is a failing of most hospitals, however, to turn down a bone marrow biopsy to the least experienced surgical interne with results that are wholly valueless as in this case.

Our sections of the bone marrow at autopsy show in all areas an extreme grade of hyperplasia which has almost completely displaced the fat even from the long bones. With careful examination it is evident that the marrow cells consist almost wholly of undifferentiated stem cells, of megaloblasts and of normoblasts. Myelocytes and leukocytes are practically absent. Such a picture could be interpreted either as a tumor-like proliferation of the red cells suppressing white cell formation by a process of displacement or as a complete failure of differentiation on the part of the white cells. Although the first possibility cannot to my mind be absolutely excluded, the picture is not that of any of the recognized types of erythroblastoma and the second alternative is the more attractive one. Is it then a leukemia? We know that leukemias can go through leukopenic stages but many of the features of leukemia are missing. There is, for instance, no trace of leukemic infiltration of the liver, spleen, or lymph nodes. The bone marrow itself, moreover, does not show the overwhelming infiltration, the almost complete displacement of red cell elements which is usual in leukemia.

Cases of this type are by no means rare and the confusion of clinical and pathologic viewpoints in regard to them is attested by the varying and paradoxical names which are given them—chronic agranulocytosis, hyperplastic type of aplastic anemia, aleukemic myelosis and aleukemic leukemia[1, 2].

My personal prejudice was strongly against regarding this case as leukemia. The process is essentially one of failure of differentiation of the white cell elements without the neoplastic overgrowth of these elements that is seen in leukemia. Very possibly it is more closely related to agranulocytosis though certainly it does not belong to the ordinary types of that syndrome. For want of a better term I shall choose the most noncommittal one available and call it aleukemic myelosis.

This is the fourth essentially similar case we have had.

Dr. Jones: Had the others a story of benzol?

Dr. Mallory: No. One had a story of having been exposed to x-rays ten years before. The patient was a janitor in a radiologist's office. The exposure was ten years back and we do not know how great it was. One was a child, and in the others I am quite sure there was no benzol story.

A Physician: What is the picture at postmortem in cases of leukemia treated with benzol?

Dr. Hunter: It wipes out the white cells in the blood.

Dr. Mallory: I do not know what it does to the marrow.

Dr. Hunter: How can we say this is not a curious stage of leukemia when we remember the case two years ago which showed a bone marrow biopsy typical of leukemia yet at postmortem two weeks later there was nothing?

Dr. Mallory: I believe that the ordinary explanation of the anemia in leukemia is that the red cell elements are displaced from the marrow by the overgrowth of white cell elements. If the leukemic cells were suddenly all to disappear from such a marrow you would be left with an aplastic marrow as in the case Dr. Hunter referred to. But in this case the red cells were not displaced, in fact they are present in increased numbers.

Dr. Jones: Is there any possibility that the large amounts of liver he had received and the nucleotide might have affected the bone marrow? He received unusually big doses.

Dr. Mallory: I think one has to admit the possibility that both of them might have influenced the picture.

Dr. Jones: It might have obscured a diagnosis otherwise recognizable.

DR. MALLORY: I think one has to admit the possibility.

DR. JONES: He had about three or four times the usual dose of liver.

DR. MALLORY: I think the observation that following the nucleotide his reticulocytes jumped four-fold is of some interest.

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1 Balbridge, C. N., and Fowler, W. M.: Aleukemic myelosis. Arch. Int. Med. 52:852, 1933.
2 Rhoads, C. P.: The modern therapy of refractory anemia. M. Clin. N. Amer. 17:1351 (March) 1934.

CASE 20362

PRESENTATION OF CASE

DR. FRANCIS T. HUNTER: A twenty-seven year old white male was admitted to the Naval Hospital, Portsmouth, New Hampshire, complaining of weakness and shortness of breath.

For about one week prior to reporting to the doctor at the naval prison, where the patient had been on inside patrol duty, he felt weak, had attacks of dizziness and frequently had to stop while walking up a flight of steps because of dyspnea. His friends remarked that he looked sallow and sickly. On the day before admission he developed a slight rise in temperature and symptoms of malaise,—lassitude and anorexia.

His family history is irrelevant.

He had had the usual childhood diseases, frequent head colds, and an occasional attack of pharyngitis. He had a bubo one year before entry, and an upper respiratory infection one month before entry which lasted for ten days.

Physical examination on admission revealed a well-nourished and developed young man weighing about 180 pounds. He was slightly jaundiced. The temperature was 101°, the pulse 115 and the respirations 20. The blood pressure was 120/70. The tonsils were slightly hypertrophied but not inflamed. Physical examination was otherwise essentially negative.

Laboratory study revealed a red blood cell count of 2,810,000, white blood cells 3,100, hemoglobin 60 per cent, color index 1.7, band forms 4, segmented 23, lymphocytes 68, and monocytes 4. The icteric index was 13. The urine was negative. A Kahn test was negative. Bile pigment was present in the feces. Gastric analysis was within normal limits.

Several days after admission he complained of painful throat. An ulcerated area, positive for Vincent's angina organisms, was present on the surface of the left tonsil. Small petechiae appeared on the hard palate. A slight cervical lymphadenopathy developed and small purpuric areas appeared over both thighs. The red blood cell count decreased to 1,820,000 and the white cells to 1,650, with a lymphocyte count of 72 per cent. Air hunger and lassitude were marked.

A transfusion of 500 cubic centimeters of blood was done. After a temporary improvement in the blood picture the anemia and the reduction in white cells progressed, reaching on the twelfth day 1,690,000 and 600 respectively. Subsequent to the transfusion the throat ulceration disappeared and remained absent. A constant elevation in afternoon temperature from 100° to 101° was present.

Liver extract by mouth and the intravenous route was used along with deep x-ray therapy to the long bones and routine symptomatic treatment. After one week on liver therapy an increase in the reticulocyte count was noted, but this was only temporary. For approximately a week, before the patient's death there was a steady rise in the number of white cells and a progressive increase in the percentage of lymphocytes. The hemoglobin and the red cell count during this time steadily dropped. On the day before death the white cell count had reached 57,650 and the red count 1,530,000. Lymphocytes by this time comprised 93 per cent of the total white cells and of these lymphocytes 50 per cent were large atypical forms. During the same period the patient complained of generalized abdominal discomfort. The small purpuric spots gradually disappeared. A gradual enlargement of lymph nodes all over the body appeared, particularly noticeable in the epitrochlear and supraclavicular regions. Dyspnea became gradually quite marked. Both ankles became swollen and pitted on pressure. The day before death an enlarged spleen extending two fingerbreadths below the costal margin was felt for the first time. During the final period the temperature varied between normal and 101°.

CLINICAL DISCUSSION

DR. HUNTER: This patient is one whom I never saw. He was never in this hospital, but the case was of such interest that I saw the blood smears before death and ventured a diagnosis. After the patient's death Dr. Hoyt of the Naval Hospital in Portsmouth was good enough to send us a report of the postmortem and blocks of tissue for microscopic examination. We have added the case to our collection for its unique features.

It is interesting to note how following transfusion the white cell count fell to 900, then to 600. The diagnosis at this time was agranulocytic angina. On the twelfth day his white cell count rose spontaneously, and at this time he showed a characteristic tumor type of lymphocytosis, so the clinical diagnosis we made was leukemia, probably of the lymphocytic type.

DR. TRACY B. MALLORY: Dr. Hunter, at what period during the time he was under observation was it first possible to be reasonably sure of the leukemia?

DR. HUNTER: We were suspicious of leukemia from the very first in a man with a white

cell count of 3,000 with twenty-nine per cent polymorphonuclears and with also a marked degree of anemia. About nine out of ten such cases are leukemias and not malignant neutropenia of unknown etiology. I think the extreme anemia is good evidence of the diagnosis in this case.

Clinical Diagnoses

? Agranulocytosis (at first).
Lymphatic leukemia (finally).

Dr. Francis T. Hunter's Diagnosis

Leukemia, probably lymphocytic in type.

Anatomic Diagnosis

Lymphatic leukemia with aleukemic stage.

Pathologic Discussion

Dr. Mallory: We have only a brief record of the gross autopsy findings.

The liver and spleen were both greatly enlarged at the time of death, the liver weighing 3400 grams and the spleen 1440 grams.

The most striking enlargement was in the kidneys. They weighed 1300 grams.

Portions of all these organs were sent down to us and I can show you some of the sections.

This first slide is the blood smear which was taken at the time when his white cell count was 600. There is only one white cell in every three or four low power fields.

The next smear was taken when his count had risen to 20,000. Even with low power you can see that the white cells are fairly numerous. The magnification is hardly enough to admit a recognition of the cells, but it is fairly obvious that they have round nuclei and the great majority certainly fall into the lymphocytic class. The number of granulocytes is not over 15 per cent, I should say.

The bone marrow is entirely characteristic of a leukemic process. The fat tissue is completely displaced by a very uniform type of cell, practically nothing but large immature lymphocytes, an occasional multinucleated cell which may be a megakaryocyte, but almost no myelocytes or islands of erythropoiesis can be found.

The liver, spleen and kidney all show marked and typical leukemic infiltration.

The final autopsy findings would leave no doubt whatever as to the diagnosis of leukemia, but at the time the patient was first seen, when the white cell count was down as low as 600, it was a case well worth speculation.

Have you anything further to add, Dr. Hunter?

Dr. Hunter: It is interesting to compare this case and the case you had two years ago in which there was a leukopenia and an anemia for many months, and which showed in a biopsy of the sternum twenty-one days before death a typical leukemia. We could never make the diagnosis from the blood smear. Yet at autopsy the marrow was typical of agranulocytosis, showing no white cells at all. This case is more or less the reverse process, the agranulocytic picture first and then the terminal leukemia, and makes an interesting comparison.

A Physician: How commonly does one see a leukocytosis in these cases?

Dr. Mallory: I have seen it in both lymphatic and myeloid leukemia that survive a stage of marked granulopenia and wind up with a terminal count which may be occasionally very high, as much as 100,000.

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JOHN KEATS AS A MEDICAL STUDENT

JOHN KEATS attended Guy's Hospital in London for a period of two years from 1814 to 1816. He had had an apprenticeship before that time with a surgeon, but we know practically nothing about this period of his life. At Guy's Hospital he listened to the lectures on anatomy and physiology given by Astley Cooper, the leading surgeon of his time. A notebook in Keats' hand was preserved for many years by his friend, Charles Wentworth Dilke, and now is kept at the Keats Museum at Hampstead, England. Some years ago, Sir William Hale-White became interested in this same notebook and gave an account of it in *Guy's Hospital Reports* for 1925. Photographic copies were made at that time and distributed to various libraries, one being sent to the Harvard College Library. It seemed best, however, in view of the importance of anything that could be found about Keats' early life, to publish this notebook and it has recently been issued in book form, edited by Maurice Buxton Forman*. Mr. Forman in a prefatory note explains that the manuscript contains notes on a series of twelve lectures on physiology, as well as notes on anatomy. The notes on physiology appear to have been taken down directly from Cooper's lectures, while the notes on anatomy probably were written up later.

It is interesting to note that young Keats, who obtained his right of qualification to practice before he reached the age of twenty-one, was an ardent and exact student of medicine. At the same time, moreover, he had written considerable poetry, some of which is of the highest standard. Although he was a brilliant medical student, who passed his examinations with ease and was sufficiently outstanding to attract the attention of a great man, Astley Cooper, at many times the student's life was turned away from, for that of the poet. It is said that he would often sit in the window-seat of the St. Thomas's Street lodgings near the Hospital, where he lived, and dreamily compose some of his finest lines. While sitting thus with a constant friend, Stephens, 'the young poet broke out with the announcement that he had composed a new line:

"A thing of beauty is a constant joy."

"What think you of that, Stephens?"

"It has the true ring, but is wanting in some way," says Stephens and goes on reading. An interval of silence and then Keats,

"A thing of beauty is a joy for ever,"

and thus was born in St. Thomas's Street one of the imperishable lines of English Poetry.' (Hale-White, p. 262.)

Thus we have added to our printed literature, of the most famous of the medical poets, an actual transcription of his school notes. An estimation of the worth of these notes has been adequately supplied by Sir William Hale-White. The man himself, and his relation to medicine in general, called forth from Sir William Osler one of the best of Osler's many papers on medical history†. That Keats was able to escape from the routine of a medical practitioner's life to give us poetry of the highest importance, written with a crystalline vision such as no one save Shakespeare has ever attained, cannot in any way be attributed to his medical training. He was, after all, only a student who had partially completed his work and was able to act as a dresser and even to do minor operations. Occasionally, after he had given up the idea of practicing, he thought of returning to his medical career, but apparently he had traveled too far

*John Keats's Anatomical and Physiological Note Book, edited by Maurice Buxton Forman. Humphrey Milford, Oxford University Press, 1934. xi + 68 pages.

†John Keats—The Apothecary Poet, by William Osler. *Johns Hopkins Hospital Bulletin*, 7: 11-16, 1896.

"in the realm of gold" to ever return to the baseness of a medical man's life. Had he done so, we might have lost some of our most perfect expressions of emotional thought.

THYMUS BOGEY MAKES FRONT PAGE

It is unfortunate, although hardly unexpected, that the dramatic possibilities of the thymus tradition should eventually bring it to the front page of the daily press, as occurred on August 6. The newspapers can hardly be criticized for making news of the sudden and tragic death of a young girl, nor for featuring a mysterious gland to which that death was attributed; the pity is that the information published abroad to a thrill loving public could not have come from a more authoritative source.

The medical profession has had to accustom itself, perhaps too complacently, to the muddling of scientific facts and the sacrifice of technical accuracy to the demands of colorful journalism. Usually this misinformation does no harm except to continue the lay ignorance of medical subjects; in the present instance much unnecessary fear and mental anguish may result and lives may even be endangered by an exaggerated idea of the risks attendant upon surgery in children.

According to the current news story, "in children up to 12 years, it has now become routine practice to x-ray for evidence of persistent thymus, because the shock of an operation may prove fatal. If the gland is evident, it can be reduced by further use of the x-ray until sufficiently small to permit operation". And again, "The thymus is unknown in adults. *Children in whom the gland persists invariably die as the result of some shock, physical or mental, between the ages of 4 or 5 and 20.*" (Italics ours.)

Fortunately, it is not so much the gland that persists as it is the misinformation that has grown up about it; a mass of misinformation that no intelligent physician should longer tolerate in view of the many careful studies that have been made and published in the last few years, totally discrediting the misconceptions that had developed about the persistent or enlarged thymus gland as a cause of sudden death in childhood and infancy.

Stories of this character are harmful. They retard medical progress, they breed doubt and lack of confidence in the lay mind, they endanger life by engendering an unnecessary fear of surgery. They should be combated vigorously by every intelligent and informed physician, who, if he needs ammunition for the assault is referred to the following publications: Boyd, Edith: *Am. J. Dis. Child.*, 43:1162, 1932; Morse, John Lovett: *Penn. M. J.*, 35:28, 1932, and *Boston M. & S. J.*, 197:1547, 1928; Garland, Joseph: *New Eng. J. Med.*, 200:59, 1929, and Capper, Aaron and Schless, Robert A.: *J. Pediatrics*, 4:573, 1934.

THE CONVENTION OF THE INTERNATIONAL ASSOCIATION OF INDUSTRIAL ACCIDENT BOARDS AND COMMISSIONS

On page 473 of this issue the program of the above designated organization appears.

Mr. Parks, the President, in a letter to Dr. William H. Robey, President of the Massachusetts Medical Society, has extended to the members of the Society an invitation to attend the convention, one session of which is devoted to the medical problems incident to the operation of industrial accident laws.

Mr. Parks also hopes that the medical profession, with ladies, will attend the banquet. This will be an interesting occasion both because of the subjects discussed and the quality of the after-dinner addresses.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Hurxthal, Lewis M. M.D. Harvard University Medical School 1923. Physician, Lahey Clinic. His subject is "Clinical Interpretation of the Electrocardiogram." Page 431. Address: 605 Commonwealth Ave., Boston.

Lord, Frederick T. A.B., M.D. Harvard University Medical School 1900. Clinical Professor of Medicine, Harvard University Medical School. Visiting Physician, Massachusetts General Hospital. President, Massachusetts Tuberculosis League. His subject is "Certain Aspects of Bronchiectasis with Special Reference to Diagnosis and Treatment." Page 437. Address: 305 Beacon Street, Boston.

Dameshek, William. M.D. Harvard University Medical School 1923. Associate Physician, Beth Israel Hospital. Physician, Boston Dispensary and Research Division, Boston State Hospital. Assistant Professor of Medicine, Tufts College Medical School. Instructor in Medicine, Harvard Medical School, Courses for Graduates. Address: 371 Commonwealth Avenue, Boston. Associated with him is

Gargill, Samuel L. M.D. Tufts College Medical School 1925. Associate Physician, Beth Israel Hospital. Instructor in Medicine, Harvard University Medical School. Address: 485 Commonwealth Avenue, Boston. Their subject is "Studies in Agranulocytosis. IV." Page 440.

GRUND, J. L. M.D. Harvard University Medical School 1921. Assistant Visiting Physician, Department of Dermatology and Syphilology, Massachusetts Memorial Hospitals. Instructor in Dermatology and Syphilology, Boston University Medical School. His subject is "Purpura Hemorrhagica with Profuse Bleeding From the Mucous Membranes Following the Treatment of Syphilis with Bismarsen." Page 443. Address: 483 Beacon Street, Boston.

FRIEDMAN, ELI. M.D. Tufts College Medical School 1913. F.A.A.P. Visiting Pediatrician to The Boston City Hospital and Beth Israel Hospital. Physician to the Prendergast Preventorium. Assistant Clinical Professor of Pediatrics, Boston University Medical School. Address: 311 Commonwealth Avenue, Boston. Associated with him is

HAWES, JOHN B., 2ND. A.B., M.D. Harvard University Medical School 1903. President, Boston Tuberculosis Association. Director, Massachusetts Tuberculosis League, National Tuberculosis Association and Rutland Cottage Sanatoria. Consultant, Beth Israel Hospital, Baptist Hospital, Jordan Hospital, Plymouth and Heywood Memorial Hospital, Gardner. Address: 11 Marlborough Street, Boston. Their subject is "Studies in Tuberculin Reactions. The Scarification, Multiple and Single Puncture Technique." Page 446.

ELEY, R. CANNON. M.D. University of Virginia Department of Medicine 1925. Instructor in Department of Pediatrics and Communicable Diseases, Harvard University Medical School. Associate Physician, Infants' and Children's Hospitals. His subject is "Spontaneous Meningeal Hemorrhage. Case Report." Page 449. Address: 319 Longwood Avenue, Boston.

EMERSON, KENDALL. A.B., M.A., M.D. Harvard University Medical School 1901. F.A.C.S. Managing Director, National Tuberculosis Association. Executive Secretary, American Public Health Association. His subject is "Essential Public Health Services." Page 451. Address: 50 West Fiftieth Street, New York City.

SEVER, JAMES WARREN. M.D. Harvard University Medical School 1901. F.A.C.S. Assistant Professor of Orthopedic Surgery, Harvard University Medical School. Associate Surgeon to Children's Hospital and Boston City Hospital. His subject is "Foreign Body in Arm." Page 456. Address: 321 Dartmouth Street, Boston.

PHILLIPS, KARL T. M.D. Tufts College Medical School 1919. F.A.C.S. Adjunct Surgeon, Day Kimball Hospital, Putnam, Conn. His subject is "A Case of Regional Ileitis." Page 457. Address: 66 Main Street, Putnam, Connecticut.

IRWIN, RUTH E. B.A., R.N., M.A. Teachers' College, Columbia University, 1932. Westbrook Junior College 1932-. Her subject is "The Medical Secretary Curriculum at Westbrook Junior College." Page 458. Address: Westbrook Junior College, Portland, Maine.

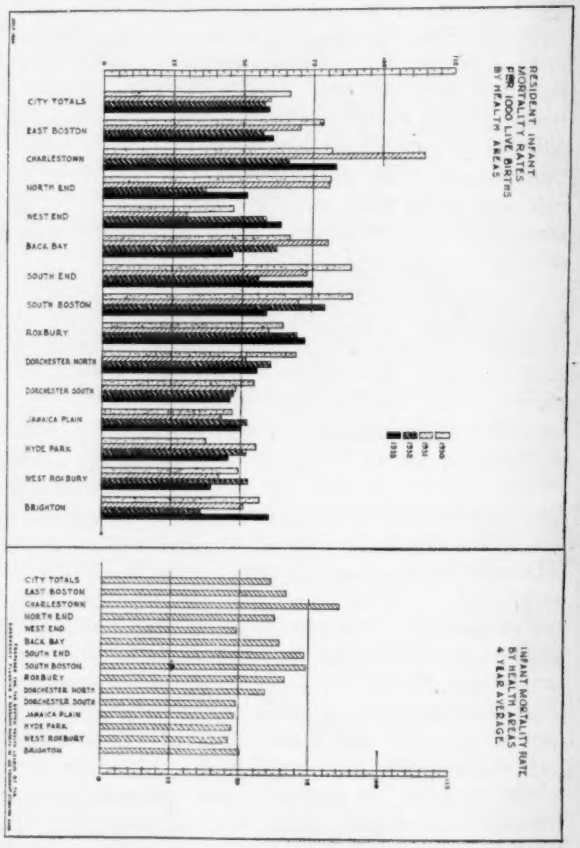
MISCELLANY

A STUDY OF INFANT MORTALITY BY HEALTH AREAS AND CENSUS TRACTS FOR 1933

PREPARED BY THE BOSTON HEALTH LEAGUE, INC.

The Boston Health League has completed its tabulation of resident births and infant deaths* for the City of Boston by health areas and census tracts for 1933, the fourth consecutive year for these tabulations.

It is interesting to note in the accompanying graph



that the West End, Dorchester South, Jamaica Plain, Hyde Park, West Roxbury and Brighton have a four year average infant mortality rate below the city average, but East Boston, Charlestown, South End, South Boston and Roxbury have an average rate consequently in excess.

In the study of infant mortality during 1933 in Charlestown and the West End, under the direction

*Births and infant deaths on which infant mortality rates were computed were furnished the Boston Health League by census tracts by the Division of Vital Statistics of the City Health Department

of a committee appointed by the Health League through the coöperation of the Boston Health Department, the Harvard School of Public Health and the Bureau of Research and Studies of the Boston Council of Social Agencies, an intensive study was made of each infant death occurring in these districts. Sixty-seven deaths were studied intensively, forty-one in Charlestown and twenty-six in the West End, medical information being obtained by a physician and social information by the Bureau of Research and Studies. "Unfortunately the number of deaths was too small to produce conclusive results. Perhaps if it had been larger it would have been equally negative, as no clear-cut trends toward definite causes were discovered in either group.

"At the outset there were those who felt that the different rates might be due to the radically different national complexion of the two districts. Nothing came out of the findings to show that this opinion had any sure grounding. No evidence was obtained to show that such factors as economic stringency, as evidenced by low incomes or relief, crowded housing conditions, or the education of the mother as bearing upon the intelligence of child care, influenced the death rate.

"Nuances of dissimilarities cropped up here and there. For example, there seemed to be a slightly greater tendency for the mothers in the West End to make use of health facilities. The five babies born at home in the West End were delivered by district doctors from the Boston Lying-In Hospital; whereas in Charlestown it was more usual, under like circumstances, to call in private physicians. Mothers in Charlestown were ever so slightly older and had undergone more pregnancies than had those in the West End. Deaths from pneumonia and still births were slightly more usual in Charlestown.

"Those responsible for the collection of the medical and social data are unwilling to attribute the difference of rate to any one cause. It is felt, however, by those on the committee, that if the material points to any one thing, it is to a need for the strengthening of medical care in Charlestown."*

The following table gives the birth rate, still births per hundred live births, and infant mortality per thousand live births for Boston, Massachusetts, and the United States:

	1930	1931	1932	1933
United States				
Birth Rate	18.9	18.	17.4	16.4
Still Births	3.9	3.9	3.8	3.7
Infant Mortality	64.6	62.	57.6	58.2
Massachusetts				
Birth Rate	17.3	16.2	15.9	14.7
Still Births	3.5	3.5	3.4	3.4
Infant Mortality	60.3	54.8	52.9	52.
Boston				
Birth Rate	17.7	16.4	16.6	15.4
Still Births	3.1	3.7	2.9	3.1
Infant Mortality	67.	60.	57.7	58.9

*From the report "A Study of Infant Mortality in Charlestown and the West End" published in the June-July issue of the *Boston Council of Social Agencies Bulletin.*

The still birth rate for Boston compares favorably with the state and country as a whole. Infant mortality for the city is higher for each of these four years than the state rate, but is nearly the same as that for the entire country. The city birth rate is higher during this period than the state birth rate, although lower than the rate for the United States.

BOSTON HEALTH LEAGUE

RESIDENT BIRTHS, STILL BIRTHS, INFANT DEATHS AND INFANT MORTALITY RATES FOR 1933 DISTRIBUTED BY HEALTH AREAS AND CENSUS TRACTS*

Census Tract	Births	Deaths Under One Year	Infant Mortality Rate	Still Births	First Day	First Month	After First Month
City Totals	12,179	717	58.9	361	172	314	231
			East Boston				
A-1	78	5	64.1	3	2	2	1
A-2	83	6	72.3	3	2	1	3
A-3	158	6	38.	7	1	4	1
A-4	92	6	65.2	6	2	2	2
A-5	139	8	57.5	3	1	6	1
A-6	114	9	78.9	5	2	5	2
B-1	61	4	65.6	3	—	3	1
B-2	88	4	45.4	5	2	—	2
B-3	78	1	12.8	4	1	—	—
B-4	75	3	40.	2	—	2	1
B-5	160	16	100.	3	7	7	2
	1,126	68	60.4	44	20	32	16
			Charlestown				
C-1	40	6	150.	4	1	3	2
C-2	68	4	58.9	5	2	—	2
C-3	89	5	56.2	7	1	3	1
D-1	27	7	259.3	1	3	2	2
D-2	22	2	90.9	1	—	1	1
D-3	38	2	52.6	—	—	1	1
D-4	55	1	18.2	2	—	1	—
E-1	80	10	125.	1	2	3	5
E-2	52	2	38.5	—	1	1	—
	471	39	82.8	21	10	15	14
			North End				
F-1	101	7	69.3	1	1	3	3
F-2	95	4	42.1	1	3	1	—
F-3	18	1	55.5	3	1	—	—
F-4	115	4	34.8	1	—	1	3
F-5	74	4	54.	—	1	3	—
F-6	8	1	125.	—	—	1	—
	411	21	51.1	6	6	9	6
			West End				
H-1	119	10	84.	4	3	5	2
H-2	51	4	78.4	1	1	2	1

H-3	33	3	90.9	1	—	1	2
H-4	**84**	**4**	**47.6**	**1**	**—**	**1**	**3**
K-1	58	3	51.7	1	2	1	—
K-2	33	—	—	—	—	—	—
	378	24	63.4	8	6	10	8

Back Bay

K-3	10	—	—	—	—	—	—
K-4	110	5	45.4	3	2	1	2
K-5	14	—	—	1	—	—	—
J-3	18	—	—	—	—	—	—
J-4	18	—	—	—	—	—	—
J-5	72	2	27.8	3	1	1	—
S-1	62	7	112.9	5	1	3	3
	304	14	46.	12	4	5	5

South End

G-1	44	—	—	—	—	—	—
G-2	32	2	62.5	4	—	2	—
G-3	4	—	—	1	—	—	—
G-4	10	—	—	—	—	—	—
I-1	49	2	40.8	—	1	—	1
I-2	100	5	50.	1	—	3	2
I-3	78	8	102.6	8	1	4	3
I-4	71	4	56.3	2	1	2	1
J-1	41	1	24.4	—	—	1	—
J-2	34	1	29.4	1	—	—	1
L-1	42	5	119.	1	1	3	1
L-2	70	6	85.7	2	—	4	2
L-3	63	12	190.	1	6	2	4
L-4	38	2	52.6	2	1	—	1
L-5	55	3	54.5	—	—	2	1
L-6	48	7	145.8	1	—	3	4
Q-1	34	3	88.2	2	3	—	—
	813	61	75.	26	14	26	21

South Boston

M-1	48	1	20.8	—	—	1	—
M-2	76	4	52.6	2	—	2	2
M-3	60	1	16.7	—	—	1	—
M-4	50	5	100.	1	—	5	—
N-1	156	8	51.3	4	2	3	3
N-2	77	4	51.9	2	1	1	2
N-3	61	2	32.8	1	—	1	1
N-4	107	6	56.1	5	—	5	1
O-1	145	8	55.2	1	3	3	2
O-2	88	6	68.2	4	1	4	1
O-3	86	8	93.	1	2	5	1
O-4	34	2	58.9	1	—	—	2
P-1	93	8	86.	2	1	4	3
	1,081	63	58.3	24	10	35	18

Roxbury

Q-2	106	14	132.1	2	1	6	7
Q-3	61	1	16.4	2	1	—	—
Q-4	76	6	78.9	4	3	2	1
R-1	132	17	128.8	4	3	7	7
R-2	84	6	71.4	1	1	3	2
R-3	76	6	78.9	2	—	2	4
S-2	39	5	128.2	5	—	3	2
S-3	119	7	58.8	5	3	2	2
S-4	68	6	88.2	3	—	3	3
S-5	88	—	—	6	—	—	—
S-6	47	4	85.1	4	—	1	3
U-1	119	12	100.8	4	1	7	4
U-2	105	10	95.2	7	—	5	5
U-3	86	9	104.6	1	1	5	3
U-4	119	7	58.8	1	3	—	4
U-5	137	7	51.1	3	2	1	4
U-6	181	8	44.2	1	1	7	—
V-1	87	4	46.	—	3	1	—
V-2	117	5	42.7	—	3	1	1
	1,847	134	72.5	55	26	56	52

Dorchester North

P-2	89	4	44.9	—	2	1	1
P-3	66	6	90.9	3	—	4	2
P-4	78	3	38.5	3	—	1	2
P-5	77	3	39.	—	2	1	—
P-6	74	4	54.	2	—	3	1
Q-5	136	10	73.5	6	—	5	5
T-1	80	3	37.5	2	—	2	1
T-2	124	6	48.4	4	—	5	1
T-3	141	10	70.9	3	3	4	3
T-4	146	15	102.7	5	6	5	4
T-5	162	8	49.4	5	1	5	2
T-6	85	3	35.3	8	1	1	1
T-7	181	6	33.1	4	2	1	3
T-8	160	10	62.5	3	4	3	3
T-9	87	5	57.5	5	1	1	3
T-10	47	2	42.5	1	1	1	—
X-1	148	6	40.5	5	2	1	3
	1,881	104	55.3	59	25	44	35

Dorchester South

X-2	172	5	29.1	3	—	1	4
X-3	199	13	65.3	5	5	5	3
X-4	187	12	64.2	6	1	7	4
X-5	256	11	43.	3	5	3	3
X-6	308	11	35.7	11	5	1	5
	1,122	52	46.3	28	16	17	19

Jamaica Plain

V-3	79	7	88.6	2	—	5	2
V-4	74	4	54.	2	1	2	1
V-5	108	8	74.1	3	2	2	4
V-6	147	7	47.6	4	1	5	1
W-1	198	7	35.3	5	3	3	1
W-2	105	3	28.6	4	1	1	1
	711	36	50.6	20	8	18	10

Hyde Park

Z-1	273	14	51.3	8	2	6	6
Z-2	141	5	35.5	4	2	2	1
	414	19	45.9	12	4	8	7

			West Roxbury				
W-3	135	5	37.	5	2	2	1
W-4	184	9	48.9	5	—	7	2
W-5	141	6	42.5	5	2	4	—
W-6	295	10	33.9	9	—	6	4
	755	30	39.7	24	4	19	7
			Brighton				
Y-1	109	5	45.9	5	3	—	2
Y-2	111	15	135.1	2	4	5	6
Y-3	225	14	62.2	2	3	8	3
Y-4	120	7	58.3	5	1	4	2
Y-5	300	11	36.7	8	8	3	—
	865	52	60.1	22	19	20	13

*Births and Infant Deaths for 1933 by Census Tracts were furnished the Health League by the Division of Vital Statistics of the Boston Health Department for this table. All births have been allocated to the residence of the mother, and all infant deaths to the residence of the decedent by the health department.

ELECTIONS TO THE EXECUTIVE COMMITTEE OF THE NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

The election of Mason H. Bigelow and George C. Clark, both of New York City, to the Executive Committee of the National Society for the Prevention of Blindness, is announced by Lewis H. Carris, Managing Director. Mr. Clark has also been selected by the Board of Directors to be Treasurer of the Society, succeeding the late George Blagden.

Dr. Edward Jackson, of Denver, Colo., Consulting Editor of the *American Journal of Ophthalmology*, has accepted membership on the Society's Board of Directors; Dr. Percy W. Cobb, Associate Professor of Applied Biophysics in Ophthalmology in the Medical School of Washington University, St. Louis, Mo., has joined the Board of Editors of the Society's quarterly journal, *The Sight-Saving Review*.

THE PHYSICIAN'S PART

Falling death rates during the depression are declared by high medical authority, not a sufficient cause for optimism. Death rates are not a "sensitive index of ill-health and do not promptly measure decreased resistance to disease". We still have to face the full impact of depression on vitality. But one saving factor is to be reckoned on in bad times as well as in good — the service of the physician. This is too little appreciated. The *London Times* recently made a special plea for the payment of doctors' bills by those who have had the benefit of their attention. Even when the physician is not paid because of the poverty of his patients, his contribution to public health should at least have its due credit.

The doctor is in a peculiarly difficult position. The sick in their need cannot be expected not to call a doctor and the doctor cannot decline to respond unless he is paid in cash or has the assurance of prompt payment. He is not in the category of the merchant or the worker under salary or wage. He is under the compulsion of his skill — as well as of his own humane feeling — to answer every call. This is part of the definition of his occupation. It would bring his profession into odium as it would the individual if he were to refuse to go to the patient though there were no certainty of compensation. As a matter of fact, much of the average physician's skill is given with certainty that there will be no payment except gratitude — and he cannot always be sure of that.

Many excuse their delay in paying the doctor, or their complete neglect of their obligation to him, by pleading their own limited means; but it is at best selfishness, or at worst something one hesitates to say, not to economize in spending for things not necessary, in order to meet a doctor's bill. As it is, the physician sets an example of unselfish service to other professions and even to trade. Without his indispensable skill freely or generously given, the depression would be far worse than it has been. That the death rate is not higher is in part due to that service seldom if ever paid in full. It is still much as it was in other times:

> God and the Doctor we alike adore
> But only when in danger, not before.
> The danger o'er, both are alike requited—
> God is forgotten and the Doctor slighted.

—Editorial in the *N. Y. Times*, August 31, 1934.

CORRESPONDENCE

THE COSTS OF MEDICAL CARE

Editor, *New England Journal of Medicine*,

Dr. Frothingham's paper on "Payment for Best Medical Care" is most timely, and inasmuch as he has not touched upon all phases of the question, I beg for a little space in which to comment.

In the beginning let me say that after forty-nine years of practice, both as a general practitioner and as a specialist, I have reached the unalterable opinion that a few years of general practice are indispensable in the training of the specialist. It is too often true that the family doctor becomes simply a "clearing house," and too often he finds himself confronted by the man who has spent his all upon experts. (Not chiropractors.) The maxim "soak the rich" was known to doctors long before the tax-gatherer discovered it, but unfortunately the rich soak themselves, as Dr. Frothingham has pointed out, especially in small centers, so they are of no value to the local doctor, nor do they contribute locally, even vicariously, to the care of the poor.

The family physician takes a case with no expectation of sending it to a specialist. He has the usual laboratory tests made, including x-ray plates, and finding himself unable to make a diagnosis sends his patient to an expert or to some big medical center. He sends a letter with the result of

tests, and the sufferer hands over the x-ray plates. All these things are immediately scrapped and a new set of tests made on a higher standard of fees. Patients complain most bitterly of this expense and they have a right to complain. It should not be necessary.

Recently a woman went to a center for observation. She had had a Kahn, a Widal, a complete blood count, a urinalysis and a blood sugar made. These were immediately done again with exactly the same results. Again, a woman came in to one of our technicians here the other day for a metabolism test. The result was a minus 36. She remarked, "Well, not much different from my last, a minus 38, but I certainly appreciate the difference in the fees—$5 against $25." These are cited to show that accurate work is done in small centers. Perhaps the patient brings a ray showing an apical abscess. Another ray is taken to confirm the obvious and the patient is advised that only a city dentist can treat that tooth successfully. We have some really good surgeon-dentists in the small centers.

I sometimes wonder if hospitals are not a bit leisurely in the treatment of inmates. The patient through years of denial has saved a little fund for this emergency, and as he lies there and realizes that it is fast vanishing in hospital fees, his state of mind militates against his cure.

In conclusion let me say that this is written without hostile animus toward anyone. I realize that "We is all bruders in dat great fraternity—." I am fully aware that the overhead is greater in cities than in small centers and consequently fees must be higher, but there are leaks in the system which should be stopped if we are honest in our contention that patients should receive the best medical care for that which they can afford to pay.

And the family doctor? Well, let us remember the woman in the Bible who spent her all upon physicians and finally came to One who cured her for nothing.

LOUIS W. FLANDERS, M.D.

Dover, N. H., August 31, 1934.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
August 28, 1934.

Managing Editor,
The *New England Journal of Medicine,*
Dear Doctor:

In addition to the articles enumerated in our letter of July 28 the following have been accepted:

Abbott Laboratories
- Ophthalmic Ointment Butyn 2% and Metaphen 1:10,000

Don Baxter Intravenous Products Corporation
- Dextrose Solutions 2½%, 5%, 7½%, 10% and 25% in Half-Size Vacoliter Containers
- Dextrose 2½%, 5%, 7½% and 10% in Physiological Sodium Chloride Solution in Half-Size Vacoliter Containers

Cheplin Biological Laboratories, Inc.
- Cheplin's Ampules Dextrose (d-Glucose) U.S.P., 50%, 50 cc. (Buffered)
- Cheplin's Ampules Dextrose (d-Glucose) U.S.P., 50%, 50 cc. (Unbuffered)
- Cheplin's Ampules Dextrose (d-Glucose) U.S.P., 10 Gm., 20 cc. (Buffered)
- Cheplin's Ampules Dextrose (d-Glucose) U.S.P., 50 Gm., 100 cc. (Buffered)
- Cheplin's Ampules Dextrose (d-Glucose) U.S.P., 50 Gm., 100 cc. (Unbuffered)

Eli Lilly & Company
- Diphtheria Toxoid, Alum Precipitated (Refined)—Lilly

Parke, Davis & Co.
- Normal Horse Serum—P.D. & Co., 1 cc. rubber stoppered vial packages
- Rabies Vaccine (Cumming) seven vial packages
- Scarlet Fever Streptococcus Toxin for the Skin Test—P.D. & Co., one 10 cc. vial package

Reinschild Chemical Co.
- Agar-Agar Shreds

Frederick Stearns & Co.
- Neo-Synephrin Hydrochloride Emulsion (Aromatic)
- Procaine-Neo-Synephrin Hydrochloride Hypodermic Tablets

E. R. Squibb & Sons
- Refined Diphtheria Toxoid Alum Precipitated—Squibb, ten 0.5 cc. vial packages

The following product has been accepted for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Nonofficial Remedies, 1934, p. 439):

Don Baxter Intravenous Products Corporation
- Physiological Sodium Chloride Solution in Half-Size Vacoliter Containers

Yours very truly,

PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

RECENT DEATHS

CHISMAN — EUDORA PIERCE (HIGGINS) CHISMAN, M.D., wife of Mr. John Chisman of Boston, Massachusetts, died at the Palmer Memorial Hospital, Boston, August 22, 1934. As Miss Eudora Pierce, M.D., she practiced medicine in Brooklyn, New York, for several years as a consultant in diseases of women. She retired from practice to marry Mr. Lewis E. Higgins of Taunton, Massachusetts, who died several years ago. Later she married Mr. Chisman, who survives her.

She was born in 1864 and graduated from the Woman's Medical College of the New York Infirmary for Women and Children, New York, in 1894. She

joined the Massachusetts Medical Society in 1908, then living in Taunton, Massachusetts. She is survived by two brothers, H. Gordon Pierce of White Plains, New York, and Dr. Hayden Pierce of New York City.

POFCHER—Elias Harry Pofcher, M.D., living at 4 Ruth Street, and having an office at 390 Main Street, Worcester, Massachusetts, died at the Beth Israel Hospital, Boston, Massachusetts, August 28, 1934.

Dr. Pofcher was born in 1881 and graduated from the Tufts College Medical School in 1904. He then began practice in Worcester and in 1907 joined the Massachusetts Medical Society. He became interested in the practice of law and after graduating from the Boston University Law School in 1915, combined the practice of law with that of medicine, gradually relinquishing the latter.

He was interested in civic affairs having served the city as councilman, president of the library trustees, trustee of the City Hospital, and was a member of the Board of Aldermen at the time of his death.

He is survived by his widow, and two children.

FISHER—Irving Jewell Fisher, M.D., of 79 Chestnut Street, West Newton, Massachusetts, died August 30, 1934, at Liverpool, England, following an operation for an acute illness. In company with Mrs. Fisher and two children, Dr. Fisher had been touring the Continent and England.

He was born in 1877, graduated from the Harvard University Medical School in 1900, and joined the Massachusetts Medical Society in 1902. He was a member of the staff of the Newton Hospital and of the executive committee of the trustees.

NOTICES

ANNOUNCEMENTS

Charles W. Milliken, M.D., of 57 Cedar Street, Fairhaven, Massachusetts, has been appointed full-time Director of Health and Hygiene in the New Bedford Public Schools.

John A. Foley, M.D., Joseph H. Shortell, M.D., and Martin F. Crotty, M.D., wish to announce that on and after September 1, 1934, they will be located in new offices at the Hotel Braemore, 464 Commonwealth Avenue. Telephone 4600.

REMOVAL

Charles W. McClure, M.D., wishes to announce the removal of his office to 406 Marlborough Street, Boston, Massachusetts. Telephone Commonwealth 0810.

NOTICES OF MEETINGS

THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES

The Association of Military Surgeons of the United States will hold its 42nd Annual Convention at Field Medical Service School, U. S. Army, Carlisle, Pa., October 8, 9 and 10.

The meetings will be of a practical nature demonstrating the latest Field Medical Equipment for the treatment of the sick and wounded and their evacuation from the field of battle; also field sanitary appliances.

The Association extends to the members of the Massachusetts Medical Society a cordial invitation to attend these meetings.

H. L. Gilchrist,
Major General U. S. Army, Ret.,
President.

WORCESTER DISTRICT MEDICAL SOCIETY

The first Fall Meeting of the Worcester District of the Massachusetts State Medical Society will be held at the Worcester County Sanatorium on Wednesday evening, September 12. Dr. Edson W. Glidden, Superintendent of the Sanatorium, will conduct members of the society through the new buildings of the sanatorium after which the institution will serve a buffet supper to the members of the society.

The Business and Scientific Program will open at 7:30 in the Nurses' Home. The scientific paper will be presented by Dr. Francis T. Hunter of the Massachusetts General Hospital of Boston. Dr. Hunter will talk on his experiences in the field of Hematology.

Erwin C. Miller, M.D., *Secretary.*

THE CONVENTION OF THE INTERNATIONAL ASSOCIATION OF INDUSTRIAL ACCIDENT BOARDS AND COMMISSIONS

The convention will be held in Boston at the Hotel Statler from September 24 to September 28.

PROGRAM

Monday, September 24

2:00 P.M.

Chairman: G. Clay Baker, Chairman, Commission of Labor and Industry of Kansas.

Effect of N.R.A. Codes on Workmen's Compensation Administration:

A. Does the Federal Emergency Relief Program Place the Responsibility for Compensation on the Locality, or Should the Federal Government Assume This Obligation? Thomas M. Gregory, Chairman, Department of Industrial Relations of Ohio.

B. Are Beneficiaries Under the Made Work or Relief Program Employees Within the Meaning of the Workmen's Compensation Act? If so, Employees of Whom? Hal M. Stanley, Chairman, Department of Industrial Relations of Georgia.

American Marriage Table, Swen Kjaer, United States Bureau of Labor Statistics.

Discussion.

7:00 P.M.
Banquet—Statler Hotel

Chairman: Joseph A. Parks, President, I.A.I.A.B.C.

Toastmaster: Joseph A. Parks, Chairman, Department of Industrial Accidents of Massachusetts.

Address of Welcome, Hon. Joseph B. Ely, Governor of Massachusetts.

Address of Welcome, Hon. Frederick W. Mansfield, Mayor of Boston.

Response, Parke P. Deans, Department of Workmen's Compensation, Industrial Commission of Virginia.

Address, Hon. Frances Perkins, Secretary of Labor.

Address, Hon. David I. Walsh, United States Senator from Massachusetts.

TUESDAY, SEPTEMBER 25

9:00 A.M.
Section A
Statler Hotel

Problems of Exclusive State Fund Jurisdictions.

Chairman, Fred W. Armstrong, Vice-Chairman, Workmen's Compensation Board of Nova Scotia.

Round Table Discussion.

Section B

Problems of Private Insurance Carrier States and Competitive State Jurisdictions.

Chairman: Peter J. Angsten, Chairman, Industrial Commission of Illinois.

Round Table Discussion.

2:00 P.M.

Chairman: G. W. Suppiger, Chairman, Industrial Accident Board of Idaho.

Control of Medical and Hospital Fees: Why and How, John C. Root, Industrial Commission of North Carolina.

Discussion: Mrs. Emma S. Tousant, Member, Department of Industrial Accidents of Massachusetts; E. B. Patton, Department of Labor of New York.

Shall the Employee Have a Right to Select His Own Physician? James J. Donohue, Board of Compensation Commissioners of Connecticut.

Discussion: William M. Knerr, Chairman, Industrial Commission of Utah.

Merit Rating, on Incentive for Accident Prevention, Evan I. Evans, Department of Industrial Relations of Ohio.

Discussion: Voyta Wrabets, Chairman, Industrial Commission of Wisconsin.

WEDNESDAY, SEPTEMBER 26

9:00 A.M.
At the Massachusetts General Hospital

Chairman: Dr. Henry C. Marble, Surgeon, Massachusetts General Hospital, Boston, Mass.

Introduction of Dr. George H. Bigelow, Director, Massachusetts General Hospital.

Treatment and Prognosis in Burns, Dr. Henry C. Marble, Surgeon, Massachusetts General Hospital.

Peripheral Vascular Lesions of the Lower Extremities, Dr. Arthur W. Allen.

Fractures of the Spine, Dr. William A. Rogers.

Industrial Poisonings, Dr. Joseph Aub.

Motion Picture: First Operation Under Ether.

12:00 M.
Statler Hotel

Chairman, Dr. Joseph H. Shortell, Surgeon, Boston City Hospital.

Study of the Cause of Increasing Disabilities in Fracture Cases, Dr. William F. Dolan, Clinic, Employers' Liability Assurance Corporation, Ltd.

Discussion: Dr. George W. Morse, Medical Director, Liberty Mutual Insurance Company, Boston, Mass.

Shortening the Period of Disability in Fractures of the Hip, Intracapsular Type, Dr. Arial W. George.

Discussion: Dr. Joseph H. Fay.

2:15 P.M.
Statler Hotel

Chairman: Dr. Francis D. Donoghue, Medical Adviser, Department of Industrial Accidents of Massachusetts; Chairman of the Medical Committee, I.A.I.A.B.C.

Low Back Strains and Their Treatment, Dr. Joel E. Goldthwait.

Discussion: Dr. James Warren Sever, Impartial Examiner, Department of Industrial Accidents of Massachusetts.

The Crippled Shoulder, Dr. E. Amory Codman, Consulting Surgeon, Massachusetts General Hospital; Impartial Examiner, Department of Industrial Accidents of Massachusetts.

Curing the Crippled Hand, Dr. William Edward Browne, Visiting Surgeon, Carney Hospital.

Kidney and Urinary Conditions Simulating Back Trouble, Dr. Herbert H. Howard, Surgeon, Boston City Hospital.

The Effect of Work on the Diseased Heart, Dr. Cadis Phipps, Professor of Medicine, Tufts College Medical School; Impartial Examiner, Department of Industrial Accidents of Massachusetts; Visiting Physician, Boston City Hospital.

The Importance of Pathological Examinations, Dr. Timothy Leary, Medical Examiner, Suffolk County, Massachusetts; Professor Emeritus of Pathology, Tufts College Medical School.

THURSDAY, SEPTEMBER 27

9:00 A.M.

Statler Hotel

Chairman: Joseph A. Parks, President, I.A.I.A.B.C.; Chairman, Department of Industrial Accidents of Massachusetts.

Reports of Convention Committees

Auditing Committee.

Resolutions Committee.

Nominating Committee.

Election and Installation of Officers.

Special Committee on Constitutional Changes, Ethelbert Stewart, Washington, D. C.

Average Weekly Wage, Dr. Stephen B. Sweeney, Department of Labor and Industry of Pennsylvania.

2:00 P.M.

Joint Session of the International Association of Governmental Labor Officials and the I.A.I.A.B.C.

Mr. Kearns of Ohio to represent the I.A.I.A.B.C. in formulating program.

7:00 P.M.

Joint Session of the International Association of Governmental Labor Officials and the I.A.I.A.B.C.

Mr. Kearns of Ohio to represent the I.A.I.A.B.C. in formulating program.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

August 18 - September 30—Medical Study Trip to Hungary. See page 975, issue of May 10.

September 10, 11, 12, and 13—American Congress on Physical Therapy will meet in Philadelphia at the Bellevue Stratford. For details write American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, Illinois.

September 10-15—First International Congress of Electro-Radio-Biology will be held in the Doges Palace at Venice. For details address the General Secretary of the Congress, Dr. Giocondo Protti, S. Gregorio 173, Venice (Italy).

September 18, 19 and 20—Tenth Clinical Congress of the Connecticut State Medical Society. See page 287, issue of August 9.

September 24-28—Convention of the International Association of Industrial Accident Boards and Commissions. See page 473.

September 27 and 28—International Association for Preventive Pediatrics will be held at Lyons. For details address the Secretary of the I. A. P. P., 15 rue Lévrier, Geneva (Switzerland).

September 28-29—New England Surgical Society will meet at Burlington, Vermont. For details address the Secretary, Dr. John M. Birnie, 14 Chestnut Street, Springfield, Mass.

October—International Medical Postgraduate Courses. See page 335, issue of August 16.

October 7-20—Sixth Annual Medico-Military Symposium, at the Mayo Clinic. See page 382, issue of August 23.

October 8, 9 and 10—Association of Military Surgeons of the United States. See page 473.

October 15-19—Sixth Annual Session of the Association of Record Librarians of North America at Hotel Kenmore, Boston. Chairman of Committee on Arrangements—Miss Edith M. Robbins, Peter Bent Brigham Hospital, Boston.

October 15-19—American College of Surgeons at Boston. See page 424, issue of August 30.

October 17—William Harvey Society. Speaker: Dr. Emil Novak of Baltimore.

October 22 - November 2—1934 Graduate Fortnight of the New York Academy of Medicine. See page 1240, issue of June 7.

October 31 - November 2—Massachusetts State Nurses' Association, Hotel Statler, Boston. For information write Miss Helene G. Lee, R.N., 420 Boylston Street, Boston.

April 29 - May 3, 1935—The American College of Physicians will meet in Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

DISTRICT MEDICAL SOCIETY

September 12—Worcester District Medical Society. See page 473.

BOOK REVIEWS

Problèmes Actuels de Pathologie Médicale. Troisieme Série. Par A. Clerc. 338 pp. Paris: Masson et Cie. 45 fr.

This third volume of presentations of medico-pathological subjects covers a wide range of material. A comprehensive review of the rôle of the carotid body is presented by Bénard. Brulé contributes a study of colitis caused by parasitic flagellates. Lian shows a number of photocardiographic tracings and emphasizes the value of this procedure. Laroche discusses gastric syphilis, and Cathala hereditary syphilis. Other contributions are by Clerc, Aubertin, Chabrol, Chevallier, Donzelot, Moreau and Vallery-Radot.

A Textbook of Histology. By Alexander A. Maximow and William Bloom. Second Edition. 662 pp. Philadelphia and London: W. B. Saunders Company. $7.00.

A second edition of Maximow's Textbook of Histology, through the efforts of Professor Bloom and his colleagues of the University of Chicago, is now available. The original book has undergone a rather complete recasting of the text and a marked reduction in size. This was done, as stated in the preface, because "the first edition of this book was found by many teachers to be too large for beginning students of Medicine".

It is still a pretty large book, comprising some 602 pages; but since its purport obviously is to present a comprehensive treatise rather than merely a program of study, its size should not be held against it. Some of the illustrations of the former edition, moreover, have been withdrawn while several excellent new ones have been added. A select bibliography, furthermore, has been appended to each chapter.

In the recast form, three things engaged the attention of the reviewer. First, the basic vital conception of the subject matter of histology; secondly, the logical directness of style employed in the introductory paragraphs of the various chapters, especially those dealing with the blood producing tissues and the nervous system; and thirdly, the precise relevancy of discussion accorded certain involved matters, such as the reticulo-endothelial cells, the neurone and its relations, and the endocrine organs.

On these considerations alone the new edition would seem to justify itself, not only as a text for "beginning students of medicine," but for others. And by reason of its rich factual content and its discriminating presentation of data relating to a variety of histological theses, it should make a wide appeal as a general source book of information in this special field of anatomy.

The Surgical Clinics of North America. June, 1934. Volume 14—Number 3. Mayo Clinic Number. 742 pp. Philadelphia and London: W. B. Saunders Company. February 1934 to December 1934. Paper, $12.00; Cloth, $16.00 net.

As usual the issue emanating from the Mayo Clinic is replete with valuable contributions. One of the most outstanding is Balfour's discussion of palliative surgery in carcinoma of the stomach. With the aid of case reports he illustrates the value of (1) radiation, (2) local resection of a recurrent growth followed by a Polya type operation, (3) division of the stomach above an obstructing growth, closure of the lower end, and end-to-side anastomosis, (4) anterior gastro-enterostomy with enterostomy and entero-anastomosis, and (5) simple jejunostomy.

Judd throws new light on many of the problems of digestive tract surgery and lays special emphasis on the selection of the proper operation for the various types of peptic ulcer. The procedures outlined in case reports are (1) excision of gastric ulcer and gastro-enterostomy, (2) segmental resection of the stomach, (3) partial gastrectomy with posterior Polya type of anastomosis, (4) excision of duodenal cap and of the anterior two-thirds of the pyloric sphincter, (5) gastroduodenostomy, and (6) gastroenterostomy.

Resection of the presacral nerves is discussed by Craig. The technic of the operation is lucidly described and the clinical application is shown in a series of case reports in vesical dysfunction, Hirschsprung's disease, and dysmenorrhea.

These with twenty-five other clinics some of which cover several subjects make this number one of real value to any surgeon.

Modern Drug Encyclopedia and Therapeutic Guide. By Jacob Gutman. New York: Paul B. Hoeber, Inc. $7.50.

"An endeavor is here made to present without favoritism or discrimination all the non-official products—proprietary, patented, Council-Accepted, and others." With this statement from the author's foreword the reviewer agrees. Utilizing 1400 pages of fine type the author has included simple drugs, combinations, endocrine preparations, biologicals, allergens, foods and beverages.

The medical profession is faced with a paradox in this country. On the one hand we find a group of active manufacturers whose standards are, in general, highly ethical and whose research laboratories have been productive of real advances. One merely needs to mention the preparation on a commercial scale of insulin, liver and stomach extracts, ovarian and pituitary extracts, refined sera, etc., to become aware of their integral function in the medical profession.

On the other hand, unfortunately, there is the concomitant situation of a duplication and multiplicity of eponyms that, to the student, resembles the confusion of Babel, to the practitioner something generally to be evaded with sporadic exceptions, and to the specialist a good deal of profane exasperation.

One will look far and wide to find a superior example of systematization and orderly arrangement to the one devised by this author. The subject matter being such as it is, he has included practically every preparation listed by the manufacturers and distributors in the country which means numerous imported products as well. The objectivity of the work is consistent with its encyclopedic nature. It would have been more fitting to have omitted the "and Therapeutic Guide" in its title. The result is that whoever utilizes this book must approach it with a preëxisting understanding of the various preparations since there is a huge amount of trash included between its covers. This is not said in derogation of its author whose task was purely collection, description and arrangement.

By so doing he has unconsciously depicted the most pitiable and sordid aspects of commercial pharmacology as well as the results of isolated examples of brilliant research.

Précis de Sémiologie Médicale Appliquée. Par M. Chiray et P. Chêne. 603 pp. Paris: Masson et Cie, 1934. 75 fr.

This book of 608 pages is intended for beginners in medicine. The authors state in their preface that the work is incomplete in that pathogenesis, rare diseases, controversial points on various diseases, and bibliography are intentionally omitted. Stress is laid on fundamentals, and the diagnosis of common disorders. Each system, such as the gastrointestinal, circulatory, and urinary systems, is separately discussed in relation to laboratory procedures, symptoms referable to each and their clinical application, and diagnostic x-ray methods.

The book is well written and adequately fulfills the purpose for which it was intended. Illustrations are numerous and well chosen.

Essays on Chronic and Familial Syphilis. By Griffith Evans. 91 pp. Baltimore: William Wood and Company. $3.00.

Dr. Evans asks, that the diagnosis of chronic syphilis be made by reasoning from pathology and

history and not dismissed because an empirical laboratory test happens to be negative. He states that it is customary to demand that one of three standards be satisfied before diagnosing syphilis,—1. Wassermann test; 2. Demonstration of spirochetes; 3. Gross pathognomonic lesion, but he feels that there is a large number of syphilitics in whom the lesions are microscopic and infection of low grade, the Wassermann negative and spirochetes no longer demonstrable. In his opinion syphilis is the commonest single disease which calls for treatment to-day. This disease is a factor in the causation of catarrh, nervous dysphagia, dyspepsia, spastic colon, abdominal adhesions, cancer, affections of the sympathetic nervous system, etc.

He cites numerous cases to prove his points. He also has photographs of numerous tongues which in his opinion are of syphilitic origin. The author has much to say about the relationship of syphilis to cancer, believing that syphilis is a common precancerous state, and cites cases and data in support of his opinion. This little book is somewhat at variance with the opinion of many students of this disease. An enormous number of people would have the stigma of syphilis attached to them if the criteria advocated by the author proved true.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

Division of Adult Hygiene

Number 17, *Cancer Clinic Bulletin*, September 1, 1934

The attendance at the State-aided cancer clinics in 1933 was 3,921, an increase of nearly 15 per cent over the preceding year. More females attended the clinics than males, the percentages being, respectively, 61.8 and 38.2. Cancer cases comprised 24.8 per cent of the total admissions. Nine hundred and seventy-three individuals with cancer were seen. There is very little change in the percentage of individuals having cancer throughout the years in which the cancer clinics have been in operation. The highest rate in any year was 25.4 in 1930 and the lowest rate 21.0 in 1928. There was an increase of nearly 40 per cent in individuals having precancerous lesions. This is gratifying, as the removal of precancerous lesions is probably the best attack upon the disease at the present time. This year showed the highest percentage of precancerous lesions of any of the years. Only 4 per cent of the individuals coming to the clinics have no pathology. This figure is about the same as that for previous years. The median age of both the cancer patients and the total clinic attendance has remained practically constant throughout the eight years of clinic operation.

The number of cities and towns from which individuals came to the cancer clinics increased considerably in 1933 over 1932. The number of towns sending one patient only has decreased and those sending more than one have all increased.

In 1933 the Pondville Clinic had about four times as many patients as the next largest clinic and this clinic comprised over one-third of the total clinic attendance. Brockton, Lowell, Lynn and New Bedford had over 300 patients each, the Boston Dispensary and Worcester over 200, and Lawrence, Springfield and Worcester North over 100. Newton and the Berkshire clinics had less than 100 cases. The percentage of cancers varied considerably in the different clinics. Berkshire, Brockton, Lowell, Lynn, New Bedford and Worcester North had rates of less than 20 per cent while the Boston Dispensary, Lawrence and Pondville had over 30 per cent. The rates did not differ greatly from the preceding year for most clinics. A smaller percentage of precancers was found in the Berkshire clinic than in any other, while the largest percentage was found in Pondville.

It is important to determine what the optimum percentage of cancers should be in these clinics. If the figure is too small or too large, cancer phobia or lack of education is probably responsible. Inasmuch as the cases referred by physicians have higher cancer rates than others, the total percentage of cancer is not a good figure to use, as in these clinics the proportion of patients referred by physicians differs. Seven of the clinics had cancer admission rates ranging between 12.7 and 18.0 for individuals not referred by physicians; two had rates over 30; and two under 10. While it is by no means certain, it is believed that the optimum figure for individuals not referred by physicians is somewhere in the neighborhood of 15 per cent.

The median duration before the first visit by the patient to a physician shows little change between the years 1932 and 1933. However, there is a decrease of about one-half month from the average of the years 1927-1931. On the other hand, the median before the first visit to the clinic shows a duration of one month less in 1933 than in 1932 and a duration of two and one-half months less in 1933 than for the average of the years 1927-1931. Breast and skin cancers came to the clinic at an earlier period in 1933 than in the previous year.

The median duration before the first visit to a clinic for total patients has decreased in 1933 from the average of the years 1927-1932 in all groups save that in which more than one physician was consulted, which group has remained constant. In 1927-1932 the duration for breast cancer referred by one physician was 7.7 months. It is now 3.7 months. This is very encouraging, indicating that physicians are referring this type of patient at an earlier stage of the disease than in former years. A similar encouraging finding is in relation to skin cancer in which the interval has decreased from twenty-two months to thirteen months among those referred by one physician. The individuals who came in for breast cancer without seeing any physician had

practically the same duration as those who were referred by the first physician consulted. The early cases of breast cancer seen in the clinics were those sent in by physicians, with those who came of their own accord and had never seen a physician, second.

The percentage of cancer patients coming to the clinics referred by physicians has increased, and now over two-thirds of all cancer patients seen at the clinics have been referred by physicians. Those referred by newspapers have decreased. The percentage of patients with precancerous lesions referred by physicians has increased as has also the total of patients. It would appear that each year consultations are becoming a more important feature of these clinics. There was a considerable increase among the patients with both cancer, and precancerous lesions who came for "all other" reasons. This increase is largely attributed to the number of former clinic patients who returned with a new lesion.

The percentage of individuals coming to the clinics at the advice of a physician has increased and those coming because of newspaper publicity have decreased for the clinics as a whole. This decrease is noted in every clinic with the exception of Lawrence, Lynn, New Bedford and Worcester, which show increases. The Berkshire clinic shows the smallest percentage of individuals referred by physicians, and the Pondville clinic the greatest percentage. The Boston Dispensary clinic shows a large increase in the number being referred by physicians. Apparently the trend is toward more physician-referred patients to the clinics and newspaper-referred less. In the first years of the clinics about two-thirds of all cases came because of newspaper publicity. For the last two years the only clinics that approach this are the Berkshire and the Brockton clinics.

Cancer patients who came to the clinics referred by physicians comprised 68.3 per cent of all cancer cases in 1933 and 64.0 per cent in 1932. Increases were noted in all types of cancer with the exception of digestive tract and buccal cavity. The radio was almost negligible in bringing cancer patients to the clinics and the newspapers were far less important than in the earlier years of the clinics. The percentage of cases coming from past experience has increased for most of the types of cancer.

The percentage distribution of cancers in 1933 showed larger percentages of uterus and skin, with a falling off of digestive tract and breast. The other types were about the same.

The diagnoses of previous years were changed in 1933 in sixty-two cases. Forty-two of these were changed from non-malignant conditions to cancer, and twenty, which had previously been diagnosed cancer, were changed to non-cancer.

Of the total cancer patients coming to the clinics, 15 per cent had never consulted a physician, 16 per cent had consulted one or more physicians but had come of their own volition, 39 per cent had consulted one physician and were referred by him, and 29 per cent had consulted more than one physician and had been referred by the last one consulted.

The distribution of the cancer cases in the group that came directly to the clinic without consulting a physician differed radically from the other three groups. This group showed a larger percentage of skin cancers, breast cancers and buccal cavity cancers, and a much smaller percentage of all other groups. Apparently educational activities are not being appreciated so well in cancer of the uterus as in the mouth, breast and skin.

The percentage of individuals coming to the clinics who had never seen a physician prior to their clinic visit was less in 1933 than in 1932. The present figures indicate that about one-sixth of the cancers seen at our clinics make their first medical contact at that place. The percentage of individuals who had seen two or more physicians increased.

The symptoms that first brought patients to the clinics closely resembled those of previous years. It is disappointing to note that so large a percentage of the cancer patients come because of pain. There was no difference between the 1933 rate for pain and that of previous years. As pain is usually a late symptom in cancer, this would indicate that the public is not yet aroused to the importance of seeking medical advice at an early period.

Among males the percentages of operable cancer with probable cure remained constant for 1932 and 1933, and the percentages of operable cancer with possible cure showed an increase in 1933. Among females there was an increase in 1933 in the operable cancers with probable cure and a decrease in those with possible cure. Breast cancer had nearly 4 per cent more operable cancer with probable cure than in the preceding year, but cancer of the uterus showed a decrease. It is discouraging to note that only 23 per cent of the breast cancers were in the probable cure class. The optimum should be around 70 per cent. It is equally discouraging to realize that less than 7 per cent of cancers of the uterus came at an early stage of the disease.

Whereas 41 per cent of the males and 32 per cent of the females were classified as operable cancer with probable cure, there was considerable variation in the individual clinics. In the male group, Springfield, Brockton, Lynn and New Bedford listed their probable cures as over 10 per cent greater than the totals, while Lowell felt that less than 17 per cent were in that class. In the female group, Berkshire, Boston Dispensary, Brockton, Lawrence and Springfield greatly exceeded that for totals, whereas New Bedford found only about 4 per cent of their cases in this classification, Lowell 14 per cent, Worcester 16 per cent, and Pondville 30 per cent. This great discrepancy is probably due largely to different interpretations in the clinics, as it does not seem reasonable to believe that such great differences actually occurred between individuals coming to the clinics as appeared between Berkshire and New Bedford.